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CYTOLOGICAL DIFFERENCES BETWEEN NORMAL AND MALIGNANT TISSUES.[1]

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IN recent years it has been realized that the phenomenon of neoplastic growth must necessarily be attacked from the cytological standpoint because carcinogenesis is essentially concerned with the individual cell, its morphology, cytology and physiology.

While histological and pathological observations on neoplasms are now well correlated and systematized in text books, the more detailed researches of a cytological nature remain still scattered at random, such that our information on even such well known phenomena as the nuclear behaviour of neoplastic cells lies uncorrelated and incomplete.

So much is now known of the behaviour of the essential bodies or cytomicrosomes within the cells of normal animals and plants that it is becoming increasingly difficult to confine our researches to the individual components of the cell, such as the Golgi apparatus or mitochondria, rather than attempting to deal with the entire cell and its anatomy.

With neoplastic cells this individual treatment of different cell elements has in some instances been carried out exhaustively, but the work is still left uncorrelated with the researches on allied cell bodies and other cytological phenomena.

Recently cytologists have attempted not only to describe the purely morphological behaviour of the various cytomicrosomes, but also to suggest the part these bodies play in the growth, metabolism and differentiation of the animal cell. With the large amount of data concerning the physiological behaviour of neoplastic cells both *in vivo* and *in vitro* it is necessary to investigate this from a cytological point of view and at the same time to give many of these physiological phenomena, recently discovered with cancer cells, an interpretation which coincides with their cytological behaviour.

In this present communication the more important cytological observations on neoplasms are dealt with and correlated and wherever possible the phenomena, hitherto unaccounted for, are given some interpretation in the light of recent researches in cell physiology.

In order to elucidate observations which are still a matter of controversy, cytological preparations have been made of a series of neoplasms and several *in vitro* experiments have been carried out.

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

We wish to express our indebtedness to Dr. Keith Inglis, of the Sydney Hospital, for kindly supplying the human tumour material and also to Professor A. N. Burkitt for his interest and help during this investigation.

Material and Methods of Technique.

During this investigation the following neoplastic material comprising both human and rodent carcinomata and sarcomata was employed: Human adeno-carcinoma of the breast, human adeno-carcinoma of the tongue, mouse adeno-carcinoma (Twort), mouse small round celled sarcoma, Lumsden rat sarcoma, spontaneous transplantable mouse adeno-carcinoma.

The following cytological methods were used to stain mitochondria and the various cell inclusions other than the Golgi reticulum. The Altmann's acid fuchsin and picric acid technique together with Benda's modified Flemming fixative were selected because the advantages of the former, according to Lee, lie in its capacity to stain granules only that may be seen *intra vitam* and in never producing artifacts. The latter fixative gives excellent results when treated with the Heidenhain iron hæmatoxylin process after counter-staining with eosin.

Regaud's formol-bichromate and iron hæmatoxylin methods were used invariably for staining nuclear as well as mitochondrial material.

In order to obtain sections depicting the Golgi apparatus the neoplastic material was fixed either by the Bensley's acid fuchsin or else by the Champy-Kull process.

Comparative Behaviour and Morphological Structure of the Cytoplasmic Organs within the Normal and Malignant Cell.

Golgi Apparatus.

When Golgi in 1898 discovered in nerve cells that had been previously treated with the modified method of silver impregnation, the cytological component which today bears his name, the existence of this cellular structure was by no means unchallenged at first and later when the morphological independence of this intracellular organ had been fully established, it attracted the immediate attention of cytologists, with the result that it was later found that the Golgi apparatus possessed a wide distribution and occurred in cells ranging from protozoa to man and from the myxomycetes to the angiosperms. Consequently a great amount of literature has been compiled dealing with the general behaviour, morphology and function of this structure within the normal and pathological cell. It is of special interest to observe that the structure and behaviour of the Golgi reticulum during dictyokinesis varies within the different cell types and in secretory cells the topographical changes of the Golgi apparatus act as an indicator of physiological reversal in cellular polarity.

Some authors have detected changes in the Golgi bodies, in the form of an alteration in shape in

pathological conditions and consequently the comparative distribution, morphology and behaviour of the apparatus within the normal and malignant tissues during cytokinesis and in the vegetative phases of the cells composing these tissues, has become the subject of an extensive study.

Tello[1] found that the Golgi bodies composing an epithelioma of the tongue hypertrophy and later fragment and totally disappear and he has also observed an initial hypertrophy followed by a vacuolar degeneration which results in a total loss of staining reaction. Tello later confirmed these results while working upon an experimental *Kieselguhr* granuloma. About the same time Fañanás[2] while working upon caseating giant cells observed a fragmentation and migration of the apparatus within the peripheral cytoplasm which also resulted in a total loss of staining reaction. Cajal[3] similarly reported fragmentation of the Golgi bodies during autolysis in normal tissues and Cowdry[4] has depicted a disintegration of the reticulum within tissues of the normal guinea-pig following phosphorus poisoning.

Earlier Legendre[5] was able to obtain a similar fragmentation of the apparatus in normal cells comparable to that reported by these authors in malignant tissues, by means of electric stimulation of the posterior root of a lumbar ganglion of the dog, while Cajal[3] found that small injections of pilocarpine also produced a fragmentation of the reticulum within the cells of the salivary gland and pancreas of mammals. The same author,[3] while examining cytologically cells of Schwann, following section of the sciatic nerve reported atrophy of the Golgi material followed by a fragmentation.

Later Da Fano[6] noted that hypertrophy followed by a change in position and partial fragmentation of the Golgi apparatus occurred in cells of the mammary gland during pregnancy.

At the present moment little is known concerning the physiology and function of the Golgi apparatus to warrant any detailed discussion. The majority of earlier workers assumed that the reticulum plays an important rôle in secretory processes; recently Ludford[7] has brought forward evidence which suggests that a functional relationship exists between mitochondria and the Golgi apparatus, it having been shown that at the mitochondrial cytoplasmic interface synthesis by enzymes occurs. He shows further that the continual diffusion of these products into the cell protoplasm inhibits further synthesis by preventing an accumulation at the surface of the mitochondria and that these elaborated products are concentrated into droplets preliminary to their elimination at the surface of the Golgi reticulum.

It has already been shown that the behaviour of the apparatus during pathological conditions has demonstrated its sensitiveness to cellular injury and stimulation. Similarly it is of interest to report and compare the behaviour and morphological variation of the Golgi bodies within the normal and cancerous cells. Various workers have shown a distinct difference in the appearance and structure of the apparatus within the normal and malignant cells of the same growth and it was first thought that this phenomenon would act as a pathological indicator in the diagnosis of malignant disease. Unfortunately the variations met with differ within the cells of various tumour types apparently according to the metabolic processes within the cell and are by no means a specific, constant feature.

Of late Walker[8] has come forward with the suggestion that the Golgi bodies are not true independent cytological organs, but rather artifacts and that the variation in the appearance may be accounted for in the lipin content of the cell both as regards quantity and the degree of saturation. The interpretation of this author is hardly tenable, as these bodies display a constant behaviour during mitosis and have recently been depicted within living cells[9] and stained *intra vitam.*[10][11]

This present investigation has shown that in the case of human carcinoma of the tongue the Golgi reticulum is more difficult to depict within the malignant cells and can be demonstrated more readily within the cells of the stroma; in this observation we support the contentions of Tello and Fañanás.

This intracellular inclusion was detected by us only within a hypertrophied giant malignant cell (see Figure VI). The cell was in the metaphase and the chromosomes forming the equatorial plate were stained more darkly than the Golgi bodies which had broken up into several smaller bodies, and were fixed when migrating towards the centrosomes at the anterior and posterior poles of the dividing cell. A more detailed examination of the cells in the vegetative condition composing the infiltrating malignant tissues showed several small, irregular, compact, juxtanuclear bodies in the cell protoplasm which were at first difficult to distinguish owing to their inability to absorb the stain. In this respect these apparent Golgi intracellular structures resemble the phenomenon previously described by Tello and Fañanás.

It is difficult to contend that this effect is due to a faulty fixation or technique, as the other cytoplasmic organs together with the chromatin network of these cells revealed selective action for the stain. And, moreover, it is agreed by most authors that the Golgi reticulum is more difficult to demonstrate in neoplastic than in normal cells.

Ludford[12] and Da Fano[13] who have both made extensive cytological studies upon the Golgi reticulum within normal and malignant cells, demonstrated a considerable degree of morphological variation of the apparatus in various cell types and also within different kinds of tumour cells; at the same time they have depicted a resemblance in structure between the type of Golgi bodies in the malignant cells and the tissues from which the tumour has arisen. Ludford[51] while observing the

behaviour in the cytoplasmic organs in transplantable tumour cells of the mouse, has shown that the distribution of the Golgi apparatus varies within the cells of different tumours and he moreover reports a considerable variation within certain periods of growth in the cells of the same tumour.

While working upon adeno-carcinoma of the mouse (No. 27, Imperial Cancer Research Fund) this author found that the apparatus was distributed throughout the cell cytoplasm in the form of rodlets in the non-acinous areas, but where the acini occur, these rodlets were aggregated in the protoplasm at the end of the cells bordering the lumen. He further reported a constant variation of the Golgi apparatus during dictyokinesis within the cells of different tumours. In those malignant cells wherein it occurred as scattered rodlets, as in the mouse adeno-carcinoma, it was influenced by the centrosomes, whereas in normal healthy cells the Golgi bodies were attracted towards and aggregated around the microcentra during mitosis.

While examining a Jensen mouse carcinoma this author again detected a variation in the morphology of the Golgi apparatus. In this specimen it appeared to aggregate in the form of clusters of small rodlets within the rest of the cell and at the commencement of cell division these rod-like bodies were scattered throughout the general protoplasm.

While this phenomenon is being discussed, it is interesting to note that Da Fano also reports a difference in behaviour as well as in the morphology of these cytoplasmic organs within healthy and degenerating malignant cells in the same growth. This investigator further found that the Golgi apparatus in the healthy cells of a Jensen sarcoma takes up a perinuclear or juxtanuclear position, while the apparatus of the degenerating neoplastic cell becomes scattered throughout the cell protoplasm instead of being collected in one juxtanuclear position. Like Ludford he depicts a specific Golgi structure for different neoplasms.

Da Fano noted in addition the disintegration of the apparatus during spontaneous absorption with certain tumours which has a parallel in the observations reported by other workers under different pathological and physiological conditions.[13]

Chromidial Extrusion.

The process of chromidial extrusion during chromidiosis in tumour cells has previously been depicted. Ludford has reported this phenomenon in the cells of a colloidal sarcoma and he further claims that the extruded material under certain conditions transforms into melanin. We find that the presence of chromidia is confined entirely to the malignant component of the sarcomata and after a detailed examination of this material, we were unable to report the extrusion of this substance even in those cells of the stroma bordering the periphery of the infiltrating newgrowth (see Figures I and II). A cytological examination of a carcinoma demonstrated that this phenomenon was not a characteristic feature of tumour cells composing this type of neoplasm, but masses of chromidia, variable in size, were occasionally detected lying in the cytoplasm of the hypertrophied giant cells in close relation to the nuclear membrane. The behaviour of this chromidial substance within the sarcoma cells is of unusual interest, as the extruded material migrates within the cell protoplasm to the periphery of the cell where it appears to become expelled in the form of small vesicles from the cytosome of the malignant component and finally passes out into the surrounding matrix where it apparently becomes absorbed (see Figures III and IV).

This abnormal process of chromidial extrusion into the matrix which we find to be characteristic of the malignant cells of sarcomata, is in itself an indication of an abnormal cellular metabolism. Another protoplasmic constituent which stains only faintly within the sarcoma cells after treatment with the ordinary chromidial technique, is apparently that of generative chromidia or trophochromatin. This substance is generally observed to lie in the naked cytoplasm in close relation to the nucleus and an examination of the sections of sarcomata suggested that this vegetative material gradually becomes absorbed by the general protoplasm of the cell. Closer observation suggests that the extrusion of chromidia is more apparent during amitosis of the sarcoma cell (see Figures III and IV) and it is of interest to record that this process of emission occurs during a period in the cell when one would expect residual nuclear material.

This phenomenon may be interpreted in view of the hypothesis of Popoff who maintains that this is a process by which the mass relation between nucleus and cytoplasm may be restored, if for any reason the amount of chromatin relative to the protoplasm has increased. The general interpretation of this author is of special interest when it is shown that the sarcoma cells are all characterized by the process of hyperchromasy (see Figures I to IV) as will be depicted in a later portion of this communication.

Mitochondria.

Ever since Beckton[14] in 1909 demonstrated the presence of Altmann's granules (mitochondria) in nearly every variety of normal cells, but reported their absence in many malignant tumour growths, the interest in these cytoplasmic inclusions has been awakened and few of the investigators of that period contended that the absence of these granules was an important sign in the general diagnosis of malignant disease. But with an improved technique the presence of mitochondria was gradually demonstrated to be a characteristic feature common to all types of tumour cells.

Not only are these intracellular granules components of all cell types, but their presence has been detected even within certain algæ and diatoms[15] and Alexieff[16] has recently reported their presence in some of the larger forms of

bacteria. In general it may be said that their existence is universal.

The statement has frequently been made that the number of mitchondria is approximately constant within similar cells of the same species; Wakelin Barratt[17] was one of the first cytologists who contended that the number of chondriosomes (mitochondria) met with in liver cells was approximately constant.

Following this contention, other workers estimated the numbers of mitochondria in neoplastic cells and reported an increase in numbers when compared with the amount present in normal cells.

When estimating the number of these cellular components in unit areas within neoplastic cells of several kinds of tumours we found there was no indication of a constancy in the number estimated within the normal and malignant cells. It is quite evident, therefore, that the mitochondrial content cannot be employed as a basis of classification.

At the same time quantitative changes in the form of an increase in the mitochondrial content are frequently met with in cells undergoing pathological changes. This phenomenon has been confirmed by many workers, among whom are Homans[18] and Policard.[19] The former demonstrated a definite increase in the number of mitochondria in the islets of Langerhans during diabetes, while the latter succeeded in estimating the numbers of these cell components in the kidney following the administration of phloridzin.

EXPLANATION OF PLATES.

FIGURE I.

Photomicrograph of a fixed section of round celled rodent sarcoma after treatment with Regaud's formol bichromate and staining with iron hæmatoxylin, depicting the differences in staining reactions between the invading sarcomatous tissue and that of the healthy fibrous stroma. Observe hyperchromasy of sarcoma cells as well as the deeply staining cytoplasm. Note the normal chromatin structure of stromal cells, together with morphology and central position of plasmasome.

FIGURE II.

Photomicrograph taken around the periphery of invading sarcomatous tissue after treatment with Benda's modified Flemming method and staining with Heidenhain's ferric alum process. Observe the intermediate cell types between the highly differentiated hyperchromatic sarcoma cells and the normal cells of the fibrous stroma, which suggest a process of apparent cellular differentiation.

FIGURES III and IV.

Photomicrographs of small round celled sarcoma of a rodent that has been fixed with a modified Benda's solution, and stained with Heidenhain's iron hæmatoxylin process. Note the morphology of the highly differentiated sarcoma cell, also the structure of the deeply staining cytoplasm as well as the hyperchromatic nucleus. Observe the process of chromidial extrusion during chromidiosis of the tumour cell. Several cells in the centre of the field contain chromidial bodies aggregated within the cytoplasm of the peripheral cell-regions. The final emission of chromidial material into the surrounding matrix may also be detected. Several cells may be seen undergoing asymmetrical division.

FIGURE V.

Photomicrograph revealing morphological and cytological characters of stromal cells, taken around the periphery of a round celled sarcoma in a rodent. The material has been treated with the Benda method and stained with Heidenhain's ferric alum process. Several plasmasomes are depicted lying within centre of nucleoplasm and small chromioles are also detected around inner surface of nuclear membrane. Larger chromatin bodies are also seen. Several cells are undergoing mitosis; cells to the right of field are in the metaphase. Note lightly staining cell protoplasm.

FIGURE VI.

Photograph of a section of a human carcinoma of the tongue. Observe the differences in the cytological characters between the hypertrophied giant cells of invading tumour tissue and those of the connective tissue cells of stroma. Note the variation in the staining reaction between the normal and malignant tissues, the general hyperchromasy of the hypertrophied giant cells and also the plastin hypertrophy. The cell in mitosis, to the left of the field, is in the metaphase and chromosomes forming the equatorial plate are clearly shown. Note the Golgi fragments migrating towards the centrosome at the anterior pole. The material was fixed by the Bensley acid fuchsin method.

FIGURES VII and VIII.

These photomicrographs illustrate hypertrophied giant cells of a human carcinoma of the tongue. Observe the proportional increase in the volume of the protoplasm to the nucleus. The cytological differences and also the staining reactions between the tumour cells and those of the connective tissue cells of the stroma are apparent. The atypical arrangement of the chromatin, together with the plastin hypertrophy, followed by distortion of the nuclear apparatus, is clearly depicted. The atypical morphology of the darkly staining chromatin bodies is a distinctive feature common only to the cells of the invading new growth. This phenomenon is demonstrated in Figure VIII, where abnormal morphology of the hypertrophied chromatin body is most evident. This material was fixed by Regaud's formol-bichromate method and stained in Heidenhain's iron hæmatoxylin and counter-stained with eosin.

In the areas of necrosis frequently seen in cancerous tissues we have found that a diminution of mitochondria together with an increase in fat formation is a significant feature of the necrotic cell. However, a more thorough examination of the cells forming necrotic areas showed no indications of chondriolysis.

Recently while observing normal tissues undergoing cytolysis *in vitro* under unknown physiological conditions, a similar process was detected within the living degenerating cell[20] and we were able to confirm the statement, occasionally expressed by other workers, that the cell mitochondria are capable under certain conditions of hypertrophy and transformation into vesicles with pronounced fatty and lipoidal properties.

Another feature accompanying this process is that of vacuolation, a phenomenon also characteristic of the necrotic cells during early phases of autolysis which we found could be induced by employing a culture medium possessing a low hydrogen ion concentration.

These observations incidentally revealed that fat formation is an intracellular phenomenon and not an extracellular process as has been suggested by some workers.[21] It has been found that these cytological organs are extremely sensitive to any changes in the cellular metabolism and respond by changes in structure and in this respect are indicative of pathological conditions.

Recently the function of these inclusions has

attracted the attention of many workers, both cytological and biochemical, and they conclude that the behaviour of these cell components during cell metabolism lends considerable support to an enzymatic conception of mitochondrial activity.[22][23][24][25]

Melanosis or Pigment Formation.

The variable amounts of iron-free pigment, melanin, so characteristic of the melanomata, have from time to time attracted the interest of cytologists who have frequently taken the pigmented mole as a simple form of melanoma for study. Many pathologists are of the opinion that the melanin granules are derived from chromatophores whose origin is obscure, while a few histologists believe them to arise from the pigment cells of the *rete Malpighii*. Some pathologists have gone so far as to suggest that this substance is derived from the hæmoglobin of the blood, but cytologists are of the opinion that melanin is intracellular and not extracellular in origin.

Szily[26] was one of the first workers to put forward this hypothesis; while working upon melanin formation in a colloidal sarcoma he concluded that the cell nucleus gave rise to this substance. He further pointed out that melanin is produced from the chromagen bodies which are derived from the chromagen substance. Rössle[27] also maintained that melanosis is correlated with nuclear phenomena.

Cowdry contends that melanin formation is connected with enzyme activity and suggests that cell injury, due to various agencies including ultraviolet rays, may initiate a heightened activity of autolytic enzymes which gives rise to an increase in the production of chromagen carried in the blood.[28]

However, the recent *in vitro* work of Fischer[29] has demonstrated clearly that the origin of this pigment is intracellular, as melanin can be produced in cells that have been cultivated *in vitro* free from vascular control. Most recent workers in this field are of the opinion that this pigment formation in neoplastic cells is a process involving cell protein and Ludford[30] holds that melanin results from the oxidation of an "acceptor" produced by a protein disintegration under the influence of a ferment (tyrosinase). This author, while observing a melanotic sarcoma in a horse, found that pigment formation was a process giving rise to cell protein and he further showed that melanosis may occur under the influence of the Golgi apparatus in a melanotic sarcoma in man. But Fremet, Moulon and Smith are in favour of the hypothesis of a mitochondrial origin of melanin. Assuming that melanin granules arise in relation to protein, the origin of this substance from cell mitochondria appears more likely, as it has been shown previously that these cytoplasmic inclusions are the *loci* of protein and of general protoplasmic synthesis within the living cell.[31]

Nuclear Phenomena.

Von Hansemann[32] and Pianese[33] were among the first investigators who sought some cytological principle or criterion for malignancy, and during this pursuit their attention was immediately drawn to the variable abnormalities in mitosis met within those cells composing cancerous tissues. The former investigator laid such stress upon the phenomenon of atypical mitosis that he even went so far as to suggest that this process was an indication that the cells had reverted to the anaplastic condition.

About this time Boveri came forward with the suggestion that as the result of multipolar mitosis certain forces were set up within the nucleus which permitted the survival of the cell, but at the same time suppressed the mechanism governing cell division. Meanwhile other workers of this period suggested that the so-called heterotypic division of cancer cells was an important factor in the causation of cancer and it was also held that hypochromatic nuclei arose from nuclei that contained a normal number of chromosomes by asymmetrical division.

A glance at the more recent literature shows that the interpretation placed upon the abnormalities of mitosis and general differences between normal and malignant cells is today invalid, but that certain of their cytological and morphological findings have been confirmed by several recent authors.

Sarcomata.

Unlike other forms of malignant growths, the sarcomata may be regarded essentially as neoplasms composed of very highly differentiated cell types. A lack of definite arrangement of the malignant components in "organoid" form separates them readily from many epithelioid tumours, while the sharp cytological differentiation of the neoplastic cell from the normal stromal elements (see Figures I, II and V) gives at once a diagnostic character to sarcomata which is not common to the carcinomata. This highly uniform morphological and cytological alteration of the malignant components in sarcomata gives way in epithelioid neoplasms to a gradual cell transformation which has very variable origins and uncertain limits, and which frequently produces no further cytological changes in normal cells than hyperchromasy.

An examination of photomicrographs (Figures I and II) depicting a rodent small round celled sarcoma after cytological treatment, reveals at a glance the clear distinction between the neoplastic cells of the invading tumour and those of the fibrous stroma.

It is seen that the stromal cells are elongate, of very variable contours, bearing here and there small fine processes which interlace with adjacent cells, thereby giving the whole picture a sponge-like appearance. The general cytoplasm of these cells fails to stain deeply and is uniformly reticulate in nature. Few cytomicrosomes may be detected, but lipoid formation is a prevalent feature.

Further observations show that the nuclei of the stromal cells are rarely uniform in morphology, generally oval in contour and sometimes quite elongate; they may also be sharply constricted or small and rounded where mitosis has just terminated.

The nuclear membrane is clearly defined and interrupted at intervals by the presence of small intranuclear chromioles lying in the chromatin network or else around the inner surface of the nuclear membrane. The remainder of the chromatin is curiously scattered and in the vegetative condition lies at random as small, irregular, deeply-staining granules in the nucleoplasm.

The morphological together with other cytological characteristics, such as the diffuse disposal of the chromatin of the nuclei of the stromal cells, gives the whole stromal tissue a lightly staining appearance which contrasts most strongly with those of the sarcoma cells (see Figure V). The plasmasomes of the stromal cells are detected by their more or less central position in the nucleoplasm and also by their uniformly spherical contours (see Figure V).

In general appearance the sarcoma cells are frequently described as resembling mature lymphocytes, as Figures III and IV will show. The majority of these cells are oval or round in shape, but occasionally the more elongated type may be detected. The nucleus occupies nearly the whole of the cell body and the general cytoplasm invariably forms only a thin layer. The protoplasm stains much more deeply than that of the stromal cells, losing the reticular character of the former cell type and assuming a somewhat granular aspect, presumably due to the increased number of cell inclusions and the products of greater metabolic activity.

Fischer[34] while staining sarcoma cells with certain *intra vitam* stains also reported that the cytoplasm of these cells took up the stain more readily and with greater depth. Hyperchromasy of the nucleus is the most typical feature (see Figures III and IV); the nuclei of these cells almost invariably become opaque with stain owing to the remarkable increase in the chromatin content and afford the greatest possible contrast with the almost transparent nuclei of the stromal elements (see Figure V). Amitosis is frequently detected among these rapidly proliferating cells and is more often seen around the periphery of the invading sarcomatous growth.

While this phenomenon was not met with in the stromal elements adjoining the new growth, we have found that this amitotic condition which is generally asymmetrical, is a characteristic feature common to the sarcoma cells (see Figures III and IV). In discussing this nuclear phenomenon it is of special interest to observe the interpretation of Brailsford Robertson[35] who assumes that asymmetrical division plays an important rôle in the causation of cancer. He further infers that, following unequal division, the daughter cells containing the larger nucleo-cytoplasmic ratio, are unable to multiply and are overwhelmed in the end by the rapidly increasing malignant cells, which arise from the daughter cells possessing the lower nucleo-cytoplasmic ratio.

Our previous observations[36] have shown that asymmetrical division is a phenomenon rarely met with in the normal healthy cell, but may be encountered during the early stages of cytolysis *in vitro* in an unchanged medium. As the cytotoxic products, presumably the result of cellular activity, accumulate, this process becomes more frequent. These *in vitro* experiments suggest that the degenerating cell liberates a substance which brings about asymmetrical cell division. We therefore suggest the possibility that similar substances may be set free from the malignant cells during the autolysis of the necrotic elements which may stimulate atypical division. Any extensive histological examination of sarcomatous tissue shows that large necrotic areas are frequently met with.

Carcinomata.

One of the abnormal conditions frequently met with in neoplastic growth is that of cellular hypertrophy and this phenomenon is generally depicted more commonly in sections of carcinomatous tissues. A pathologist designates such cells as giant cells and Ewing[37] lays such stress upon this process that he regards the appearance of such a phenomenon as an important means of diagnosis. This author maintains that in many respects they are peculiar to each tumour type. We were unable to detect any such process after an extensive examination of fixed sections of sarcomatous tissue, but on the other hand giant islands were readily detected in a human carcinoma of the tongue as well as an adeno-carcinoma of the breast (see Figures VII and VIII). However, no cytological differences between the two hypertrophied cell types were revealed. This phenomenon is not a characteristic feature of malignant cells alone, as the cells adjoining the stroma were at times similarly affected, while the cellular stromal elements further removed from the invading zone of newgrowth appeared normal and unaffected.

It is interesting to note that Ludford[38] suggests that this cellular hypertrophy of the stromal elements may be due to the neoplastic cells releasing some growth-promoting substance which indirectly brings about the abnormal hypertrophy of the adjacent cells.

An examination of the nuclei of these giant cells (see Figures VI, VII and VIII) shows a proportional increase in the volume of protoplasm to the nucleus. The arrangement of the chromatin within the nucleus of the carcinomatous giant cells is distinctly atypical. Even during the vegetative phase of the nucleus the normal chromatin structure found in healthy non-malignant cells is met with. During this stage large, darkly stained, swollen chromatin bodies are a distinctive feature

of the nucleoplasm and invariably appear in the form of distorted crescents or batons lying more or less within the centre of the cell nucleus (see Figures VII and VIII). Small chromioles which generally arrange themselves around the inner surface of the nuclear membrane within the normal connective tissue cells of the stromal areas, are absent.

A detailed examination of carcinoma cells shows that hyperchromasy is the dominant cytological feature of these components. Another interesting cytological difference between normal and malignant cells and especially within the cells of the same growth is that of plastin hypertrophy which is always accompanied by distortion of the nucleolar bodies. This intranuclear phenomenon is more readily detected in the carcinomatous growth during the vegetative phase and the abnormal hyperchromasy of the nucleoli of the sarcoma met within our preparations, makes it impossible to detect any similar abnormal structure in the nucleolar apparatus.

Similar observations have been reported by Walker and Debaiseux[39] in which they find that the nucleoli of an epithelioma and an adenocarcinoma of the breast display irregularity in shape and are frequently multilobular and less homogeneous and compact than the plasmasome structure in normal cells of the same growth. They further observed a similar behaviour of the nucleolus in inflammatory tissues, but noticed a normal condition of this structure within the nucleoplasm of normal and healthy cells.

We have found after detailed examination of various types of carcinomata, including a human carcinoma of the tongue, adeno-carcinoma of the breast and also in mouse Twort carcinoma and in a rodent spontaneous transplantable adenocarcinoma, that the invading malignant cells are all characterized by the unusual irregular hypertrophied structure of the plasmasome body, while even cells of the adjacent stroma contain normal spherical compact nucleoli.

Our previous *in vitro* experiments have shown that a similar phenomenon may be brought about within the normal tissue cells where chromatolysis of the nucleus is accompanied by hypertrophy and distortion of the nucleolar material during the advanced stages of cellular cytolysis occurring in an unchanged culture medium.

The evidence based upon the general behaviour of the plasmasome during cellular degeneration *in vitro* suggests that it has some functional relationship to the morphology of the cell.[40]

Cellular Differentiation.

A detailed microscopical examination around the periphery of the invading sarcomatous growth of a small round celled sarcoma in a rodent revealed cell types intermediate between the highly differentiated, hyperchromatic sarcoma cells and the healthy normal cells of the fibrous stroma (see Figure II). This process was detected in all preparations after treatment with a cytological technique previously mentioned in an earlier portion of this communication.

Extensive observations strongly suggest a process of cellular differentiation as the photomicrographs Figures I and II will readily depict. A further cytological examination of these intermediate cell types shows that the cells in the process of apparent differentiation possess cytoplasm which, while staining more deeply than that of the stromal cells, still retains a somewhat coarsely reticular appearance and the general contours of this protoplasm still approximate to those of the regular stromal cells. Hyperchromasy of the nucleus has clearly commenced, but is not sufficiently advanced to render the nucleus entirely opaque (see Figure II). Instead portion of the nuclear membrane and irregular masses of chromatin may still be recognized. The cell nucleus becomes gradually more spherical and a slight reduction in the volume of the entire cell is noticeable (see Figure II) until the transformation from the larger irregular stromal cells to the distinctly smaller malignant elements is complete.

The actual process of apparent cancerization in carcinomata must be a rapid one, because intermediate stages in the differentiation of this type of neoplastic cell are not common. In fact with routine pathological treatment the carcinoma presents merely the stromal and hyperchromatic malignant cells without intermediate cell types.

It is agreed upon that nuclear behaviour is closely concerned with the vital phenomenon of differentiation and the emission of chromidial material, such as we have depicted from the nucleus into the general protoplasm, together with its final extrusion from the cell into the matrix, are very suggestive of an abnormal cellular metabolism.

While discussing this process of cellular differentiation it seems likely to us that the differences in morphological structure, such as are displayed between various cell types, are the visible expression of a variation in chemotactic phenomena. There seems little doubt that the reaction, primarily produced within the living substance which results in a gradual transformation or differentiation of the whole cell, is brought about as the result of some intrinsic chemical stimulus which may quite possibly be derived from a purely extrinsic source. Moreover this extrinsic factor is apparently able to exert its influence only upon those cells which are receptive to it by virtue of their intrinsic factors.

It would follow, therefore, that the morphological differentiation produced in living cells is possibly a secondary issue dependent upon the intrinsic process.

Farmer, Moore and Walker[41] were among the earlier workers in the field of cancer to maintain that a primary growth may originate in the first instance as the result of a change in the nature of a number of previously functional somatic cells, by the interaction of a differential process. A few

modern pathologists among whom is Kettle,[42] are also of the opinion that the malignant monocytes of sarcomata may possibly be derived from the fibrous connective tissue elements.

Our previous experiments *in vitro* upon the cell elements of sarcomata have shown that the malignant monocyte may under certain physiological conditions dedifferentiate into fibroblasts or pseudofibroblast components and may at the same time still retain their malignant characteristics. These results, as will be shown later, may be correlated with the recent work of Carrel.[43]

Explantation *in vitro* gives a much more complete picture of the detailed cytology of the malignant elements and affords opportunity to observe the transformation which the sarcoma cell undergoes in its final stages of differentiation. Three cell types after several hours' growth *in vitro* may be easily distinguished in actively proliferating explants of sarcomatous tissue. One has been shown to be non-malignant by isolation and inoculation into healthy animals and it is therefore suggested as being of stromal origin. This cell type, by virtue of its amœboid structure, has been termed cytologically a clasmatocyte or histiocyte.

The typical oval or spherical sarcoma cells which are the main cell types observed in sections of fixed sarcomatous tissue, are present in unaltered form in *in vitro* cultures, but they are surpassed in numbers by the pseudofibroblast-like elements which are the actively migrating cells of a healthy proliferating explant. This pseudofibroblast has been demonstrated to be actually a morphological variation of the round sarcoma cell and the process of its transformation into an elongated spindle-shaped element has been previously observed by us *in vivo*.[44]

This process begins with an elongation of the round cell, followed by the outgrowth of large pseudopodia and an alteration in the shape of the nucleus as well as in its other cytological characters. Further confirmation of this process of dedifferentiation of the pseudofibroblast lies in the fact that Carrel[43] has proved its malignant nature by inoculating an isolated strain of the fibroblast-like elements growing *in vitro*, into a healthy rodent and has reproduced the original tumour type.

Observations have been made on the behaviour of the infiltration of sarcomatous cells into normal tissues growing *in vitro*, by placing a small fragment of non-cancerous healthy tissue from the same host side by side with the neoplastic tissue in the same culture medium after the former has been cultivated for twenty-four hours *in vitro*. An *in vivo* examination of these living cells shows that the round monocytes first fill the area between the two explants and aggregate on the periphery of the normal tissues together with the histiocytes.

Before the round sarcoma cells penetrate this healthy tissue which is still in a compact condition, they undergo a transformation into fibroblasts which appear to be the only cell elements capable of penetrating and infiltrating healthy normal tissues *in vitro*.

Certain Physiological Differences Between Normal and Malignant Cells.

Having indicated the detailed cytological and morphological variations between cells composing normal and neoplastic tissues, it is of importance to discuss the factors responsible for atypical cytological phenomena in these tissues.

It is already known that morphological and cytological differences between the cell types are the expression of varying chemotactic principles. Hence a consideration of the physiological behaviour of normal and malignant cells under conditions in which chemotactic influences may be altered and controlled, will help to explain many of the characteristic differences between normal and neoplastic tissues.

Lambert[45] in 1912 was one of the first workers to study *in vitro* the comparative behaviour of cancer and normal cells cultivated under the same conditions. By subjecting normal fibroblasts and sarcoma cells to different degrees of temperature for certain periods, he succeeded in showing that malignant cells were more susceptible to a rise in temperature than normal cells. Following along similar lines Fisher[46] demonstrated that a malignant cell, a sarcoma, when completely isolated, was incapable of proliferating for prolonged periods *in vitro*, while a normal cell when subjected to similar treatment was able to survive. He concluded by assuming that cancer cells are capable of synthetizing protoplasm from their own serum constituents and dead muscle tissue by which they grow. He further pointed out that sarcoma cells exhibit greater mobility in tissue cultures than normal cells. While observing the comparative behaviour of normal and malignant cells in later experiments, Fisher[47] showed that carcinoma cells grow more rapidly in a medium which has been previously heated to 56° C. for one hour, but that these conditions are unfavourable to the proliferation of normal cells. He also demonstrated that cancer cells are more susceptible to high or low tensions of oxygen than normal cells and cease to grow at low oxygen tensions in which normal cells are able to survive.

When working along similar lines Wright[48] in addition showed differences between normal and malignant cells *in vitro* when subjected to varying oxygen tensions. This investigator found that the critical oxygen tension for a rat sarcoma and the mouse carcinoma is considerably lower than that of normal cells (myeloblasts).

The result of a series of experiments showed that the lowest oxygen tension at which mitosis takes place, is for the myeloblasts twelve millimetres of mercury, for sarcoma six millimetres and for carcinoma three millimetres which corresponds to 1·6% and 0·8% and 0·4% of oxygen at atmospheric pressure.

Recently Chambers and Scott[49] have suggested that a substance is produced from tumour cells during autolysis which *in vivo* brings about an appreciable increase in the growth rate of the neoplasms. These authors who carried out their

work with the Jensen rat sarcoma, further claim that this substance is a fatty, stable, chemical body and may possibly be derived from the nuclear structures.

Tissue culture experiments recently carried out by us are suggestive that tumour material when growing *in vitro*, is capable of liberating into the culture medium a growth-promoting substance. During this investigation a small round celled mouse sarcoma was employed and it was clearly demonstrated that small fragments of normal, healthy fibroblastic tissue when cultivated side by side in the same medium, together with the small fragments of tumour tissue, grew very much faster than the controls which were similar fragments of normal tissue from the same rodent grown under exactly the same conditions for variable periods.

Examination of several series of fixed *in vitro* explants showed clearly an enormous increase in the growth rate of the normal healthy fibroblastic tissue cultivated together in the same medium as the tumour material, when compared with those of the controls. We hold that these observations are suggestive that living neoplastic tissue gives off a substance containing growth-promoting properties into the culture media which is capable of giving rise to a more active cellular proliferation of normal elements.

Fischer, while experimenting with a Rous sarcoma *in vitro*, held that neither the plasma nor living tissues from the fowl which display a natural resistance to the neoplasm, were found to have the slightest inhibitory effect upon the sarcoma cells *in vitro*.[50]

We suggest that the results of this author are further indicative of the existence of the principle responsible or required for active proliferation and that this substance is manifested within the malignant tissues themselves.

Summary.

1. By a consideration of cytologically treated preparations of various human and rodent neoplasms and of recent literature the occurrence and behaviour of the Golgi apparatus, mitochondria, melanosis and chromidial extrusions have been discussed in detail.

2. An interpretation of the phenomenon of chromidial extrusion in view of the hypothesis of Popoff is suggested.

3. Recent observations upon the function of mitochondria and on their behaviour in cells undergoing cytolysis may help to account for several atypical phenomena in neoplastic cells.

4. The various theories of melanin formation are briefly treated and evidence in favour of the mitochondrial origin of melanin has been suggested.

5. Nuclear behaviour in neoplastic cells has been contrasted with the normal processes in healthy cells. The detailed cytology of sarcoma and carcinoma cells has been described with particular attention to these nuclear phenomena. Recent observations upon the behaviour of the nucleolus in normal and neoplastic cells are also discussed.

6. The phenomenon of amitosis, so typical of neoplastic cells, has been correlated with the same process occurring in cells undergoing cytolysis *in vitro* in an unchanged medium. It is suggested that the degenerating cell liberates a substance which gives rise to asymmetrical division and that the extensive necrotic areas, so frequently met with in neoplastic tissues, also liberate a similar substance which brings about amitosis in the tumour cells.

7. In sarcomata evidence of a gradual process of cellular differentiation from the normal stromal elements to the highly specialized sarcoma cells has been brought forward and an interpretation of the part played by chemotactic principles in this phenomenon has been suggested. Recent new observations on the behaviour of sarcomata *in vitro* are dealt with in support of the theories of cell differentiation.

8. A discussion is given of the more important physiological differences between normal and malignant cells cultivated *in vitro* and evidence of the existence of a growth-promoting principle in neoplastic cells is briefly summarized.

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COLOUR BLINDNESS IN SCHOOL CHILDREN.[1]

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By colour blindness I mean a condition in which there is defective discrimination of colours. It is not an indication of any defect in the optical mechanism of the eye, for the acuity of vision may be normal, although the field of vision for the different colours is restricted.

My reasons for speaking about this subject are because it is a very common condition, it is of great importance from the industrial standpoint and it forms a very interesting line of research. The subject is really quite simple in its general aspects and becomes even more simple when we realize that the majority of people are relatively colour blind to some extent, for they do not see seven distinct colours in the solar spectrum but only six.

My remarks are based on the examination and reexamination of about 20,000 school children, chiefly boys. Only on rare occasions do I have an opportunity of examining adults and consequently I have not included them in my statistics. There is no agreement among specialists as to the part of the body which is concerned in the defect. Some theories are based on defects in the retina; others declare that the occipital cortex is the site of the trouble, while still others say that it is a mental defect, the physical part of the organism not being involved. The condition often appears to be hereditary, being transmitted from a man, through his daughters, to his grandsons. Sometimes all the boys in a family are affected and in these circumstances the blindness is nearly always of the same type. Out of 42 families with a total of 93 colour blind boys in only four families with a total of nine boys were any differences found between the members; in other families some of the boys have normal colour vision, while others are colour blind. The majority of children cannot give information about the

[1] Read at a meeting of the Ear, Nose and Throat Section of the South Australian Branch of the British Medical Association on October 1, 1929.

incidence of colour blindness among their relatives, so that my statistics are very far from being complete in this respect and are, therefore, of very little value. But I have come across families in which no cases of colour blindness had previously been known; in others the father was known to be colour blind; in others it is the mother who is affected; while in still other families one or more uncles have suffered. In a few cases the paternal grandfather was known to have been colour blind.

It is a remarkable fact that the majority of colour blind boys are not aware of their defect, but a fair proportion know that they are "not good at colours," having found out by experience in pastel drawing that it is necessary to get help from their neighbours before they can select the correct pastel. This is not due to any defect in their general intelligence, for the incidence of colour blindness is approximately the same in opportunity classes for backward children, in primary schools and in high schools. But the condition attracts more attention when it occurs in bright pupils than when it is found among the less brilliant, for any defect in the former is more conspicuous than when it occurs among those who are retarded because of other physical or mental defects. As I have not tested more than two or three hundred children in opportunity classes, it is possible that I may have to change my opinion about them in the future.

Methods of Testing.

The tests which I use are: Ishihara's colour plates, the wool test, the solar spectrum.

Ishihara's Colour Plates.

Ishihara's colour plates were designed by Dr. Ishihara, Professor of Ophthalmology at the Imperial University of Tokio. They are a modification of Stilling's plates which were introduced about forty or fifty years ago. Each plate consists of a white card filled with circular coloured dots. These dots are of various colours and are so arranged that some of them form a background, while others are grouped to form numbers. This test depends on colour contrast. It is the contrast between the dots which form the number and those which form the background, that makes the number visible. Where there is no colour contrast, the number is invisible. For example, if the number 8 is made up of pink dots in the form of a 3 and the rest of the number of brown dots, the whole standing on a background of green dots, then if there is no colour contrast seen between the green and the brown dots the number will be read as 3; if the contrast can be seen, it will be read as 8. Some of the numbers are visible to a person with normal colour vision, others are invisible; but a person who is colour blind, will see numbers where we see no numbers; he will see none where they are quite plain to us and on the other plates he will see numbers which are quite different from those which we see. Altogether the book consists of thirteen plates with numbers and three other plates which I scarcely ever use. A normal person can see, but not very distinctly, the numbers which are seen by the colour blind by looking at the plates through a piece of dark blue glass. A colour blind person will in most cases see the correct numbers by looking at them through a piece of photographic red glass. Every girl and boy whom I examine, gets this test. It is simple, quick and easily understood by all children above grade II. Where they are colour blind and have any difficulty in seeing the first number (12), I turn to those plates on which we do not see any numbers (5 and 2) and they can then easily understand what is required, as these numbers are frequently the most distinct to the colour blind. The plates are not a guide to classification, for some boys can read these numbers correctly and yet see only two or three colours in the spectrum; others make many mistakes with the numbers, but see all six colours in the spectrum. Such instances, however, are too rare to detract from the value of the book as a convenient preliminary test for separating those with normal colour vision from those who are abnormal. Formerly I did not carry out any further examination in those who got seven or more out of the thirteen numbers correct, but now I examine all who make any mistakes except in the plate with the number 74. This, in my experience, is too difficult. More than 75% of school children read it incorrectly as 21, 24, 71, 91 or 94. Where it is necessary to convince the teachers or the parents that the boy can actually see numbers which are invisible to us, I get them to look at the plates through the dark blue glass; the boy is similarly convinced that his colour vision differs from that of the majority by looking at the plates through the red glass. These glasses are, of course, quite useless for the purpose of enabling him to carry out the other tests correctly. All that they do is to absorb some rays and thus change the contrast between the dots of different colours so as to make some numbers visible and others invisible.

The Wool Test.

In the wool test I use forty different colours and shades and eleven test skeins. The number of wools has varied a lot, having at one time been 180, but I have gradually eliminated those which the patients always group together, so as to avoid duplication. Each piece of wool is attached to a paper disc bearing a number so that a permanent record of the wools chosen may be obtained. This can be compared with the results of subsequent tests. The test skeins are numbered 1 to 11. They are green, pink, red, yellowish-green, yellow, orange, blue-green, blue, purple, violet and brown. These are not given in any definite order, but care is taken not to give wools whose colour in the spectrum lies adjacent to the colour which has just previously been used. My reason for doing this is that a boy who has been tested with yellow-green, number 4, would probably say that number 5, yellow, is the same colour and that he had already done this test. If he were told that it is a different colour, there would be a big possibility of mental confusion being produced and this would make the test un-

necessarily difficult. It seems to be a very simple test, but it is only necessary to try it on a few colour blind boys to see that it is not easy for them. One of their characteristics is the ease with which fatigue for colours can arise and in order to get the best results it is necessary to make the test as simple as possible. The boy is given one of these test skeins and asked to select all the pieces of wool of the same colour; it does not matter how light they are nor how dark, so long as the colour is the same. They are also asked to name the colour of the test skein. The results obtained in this test, as a rule, are such as would be expected from the spectrum as seen by the boy. Conversely, it is possible to make a fairly good forecast of what his spectrum will be by noting which colours are chosen in the wool test. In addition to the colours which are chosen, there is frequently much hesitation in making a selection. I have seen a boy pick up a green almost the same colour to me as the green test wool, examine the two very carefully, reject the green and promptly select a brown or red in its place. On other occasions I have seen them compare a red and a green for some minutes before coming to a decision. I do not impose any time limit for these tests.

The most striking results are obtained with the totally colour blind and with the two unit class, that is with those whose spectrum consists of two colours only; and, as more colours become visible in the spectrum, the results gradually approach the normal. The very dark reds, greens and browns may not be selected with any of the test colours. If questioned, the boy would probably call them black. Similarly pale pink, pale blue, white and grey may all be grouped together.

The Spectrum Test.

I use the spectrum test as the basis of classification as advocated by Dr. Edridge-Green.[1] It is simple, requiring only a prism and the sun's rays; it is quick, easily understood and the colours obtained are constant, whereas in other tests they are always liable to fade. Two kinds of spectrum are obtained, the open and the enclosed. In both cases direct sunlight is used, but in the open type the spectrum is allowed to fall on the floor, walls or other convenient object. By this means a spectrum up to 60 or 90 centimetres (two or three feet) in length can be obtained. The advantage of the open spectrum is that an elongated strip of colours can be seen in which the colours appear to a person with normal colour vision to blend with the adjacent colours, while many of the colour blind see bands of grey or white separating two or more of the colours. One drawback of the open spectrum is that the shape of the band of colour varies according to the length of the spectrum. When it is short the colours form a rectangle; but when it is long the red end is curved outwards almost like a semicircle and the violet end is drawn out into a blunt point. Except for green, each colour thus forms a curved band with much overlapping at the borders of the strip. Unless the boy is told to indicate the colours which are seen only in the middle of the band and to ignore the edges, confusion will occur. Another drawback to this type of spectrum is that the colour of the background is not always what could be desired and, since the colours which are seen depend on the colour of the background to some extent, this becomes of importance. In order to overcome this I use a prism placed inside a box which is painted black inside, the prism being fixed in such a position that the spectrum falls on the back of the box. A space is left on the top to enable the spectrum to be seen. This is what I call an enclosed spectrum; it is compact, about five centimetres (two inches) long, and can be allowed to fall on coloured paper so as to get different results. As a rule I use dull black paper. In such a spectrum grey bands, if any, are generally so narrow as to pass unnoticed unless the boy is asked if he can see them. The commonest position for such a band is between blue and green, but it may be so wide as to occupy most of the blue and a fairly large part of the green. In other cases blue and green are said to meet, but the colours appear to get so "thin" near their junction as to make them almost invisible. People with normal colour vision do not talk about "thin" colours. One boy explained what he meant by comparing each band of colour to a wedge; the thin colour is that which is seen near the apex of the wedge.

It is not possible for me to make accurate measurements of the parts of the spectrum with the apparatus at my disposal, consequently I do not make use of terms involving wave lengths, but refer to the colours only. One word of caution is necessary. In dealing with high school pupils the word "spectrum" should not be used. Most of these boys know from text books that the spectrum should consist of seven colours and if they know that they are looking at a spectrum they will divide it into seven different parts. If all that they know is that they are looking at a group of colours, they will describe what they see and are sometimes very much surprised when they discover that it is a spectrum.

One boy who was studying physics for the leaving honours examination, could see only blue and yellow in the spectrum, but knew what colours he should have seen. He admitted that the colours which he saw in the school laboratory were very indistinct, but since the other boys said that they were all right, he did not pay much attention to them.

Classification of Colour Vision.

The vast majority of people can see six distinct colours in the spectrum—violet, blue, green, yellow, orange and red. Such persons have six units of colour vision and this is adopted by Edridge-Green as the normal. Some people can see another colour, indigo, lying between violet and blue; these have seven units of colour vision and are therefore supernormal. Edridge-Green[2] states that only one person in about two or three thousand can see indigo distinctly, while Houston[3] in his book on "Light and Colour" states:

Out of 18 observers taken at random I found that three at least saw indigo as a separate colour and there was a fourth case doubtful. The others were normal. So the peculiarity appears to be fairly common.

To me it appears as a rather dark and indefinite band visible only when the spectrum is about 18 or more inches long and projected on to a dull black screen. Those who see fewer than six separate colours in the spectrum are colour blind, but the degree of colour blindness varies much in different persons and in the same person from time to time. Each colour which is seen, is described as a unit, so that we have persons whose colour-vision belongs to the 7, 6, 5, 4, 3, 2, or 1 unit class. That is, they can see that number of distinct colours in the spectrum. The totally colour blind do not see any colour in it; their spectrum is thus achromatic and they have 0 (nought) units of colour vision.

The simplest way is to start with those who see the spectrum as a whitish band. I know of only one boy in South Australia belonging to this type.

His spectrum is what he sometimes calls dark, dark white, dark grey or black at the two ends and white in the centre. The white part includes the blue, green and yellow parts of the spectrum. He is totally colour blind, that is his spectrum is achromatic. The result of his wool test can be seen from his chart, while some pastel drawings which he made, will show how much his colour sensation differs from the normal.

In the next type the spectrum should consist of one colour only. I have not yet found anyone belonging to this type.

The next or two unit type is very common. They divide the spectrum at the junction of the blue and green into two parts. Most frequently these parts are called blue and yellow, but the blue end may be described as violet, purple, green or heliotrope and the rest of the spectrum as green, yellow, orange, red or brown. Out of 171 children with two units of colour vision, 107 or more than 62% described the colours as blue and yellow. The important thing is the division of the spectrum into two colours only. This type comprises about 30% of the total.

The next type can see three colours in the spectrum. I have included among the three unit class those who can see only blue and yellow in the spectrum, but who divide the blue into dark blue and light blue. They insist that these colours are quite distinct in appearance, but that they are both blue. They form about 5% of those with three units of colour vision. In order to get three different colours in the spectrum we have to add an additional colour to the spectrum seen by the two unit class. This can be done by separating a part from one of the ends, thus giving either violet, blue and yellow or blue, yellow and red or by taking a portion of the yellow next the blue and calling it green, thus getting a blue, green, yellow spectrum. The blue, yellow, red type is most common. Out of 161 three unit children they comprise about 55%, blue, green, red coming next with 18%. The three unit group is approximately as common as the two unit group, forming 27% of the total; between them they comprise about 60% of the total. The four unit type is formed by adding another colour to one of the three unit types. They form about 25% of the total. The addition of still another colour produces the five unit type who amount to 16%. The last colour required to complete the normal spectrum is as a rule orange. So far I have made 597 examinations with the spectrum of those who failed to pass Ishihara's test.

Besides these departures from the normal there are many variations. Some of the boys cannot see the extreme ends of the spectrum, but it is not possible to make accurate measurements with the instruments in my possession. Some boys see a grey or white band lying between the blue and the yellow. Out of 32 who have described such bands, 22 saw them in this position. This may occupy part of the green, part of the blue or rarely form a wide band where we see both blue and green. Such bands are found in other parts of the spectrum as well, but these are very rare. Two boys out of 597 could see a grey band at the violet end and one boy can see a band between orange and red, four can see them between blue and green, two between green and red and two between yellow and red. Such bands are important since any colour which lies in the part of the spectrum occupied by the band, will be called grey or white and will be matched with grey, white and other colours which are thought to be grey or white, such as pale blue or green.

A certain amount of dye can be added to a white object before it becomes definitely coloured. These boys require a greater concentration of colour before it will become visible to them. This applies not only to the addition of a colour to white, but to the mixture of two colours where one is very much stronger than the other. Purple may be called blue if it is composed of a lot of blue and not much red; puce becomes red since the blue component is not noticed; greenish-yellow becomes yellow; light pink, light blue or light green becomes white or grey and so on. Where a very dark colour is used, the effect is similar to that which would be obtained by the addition of some colour to black. In such a case dark red, dark green, dark blue or dark violet will be called black since the colour is not sufficiently conspicuous to become visible to these boys. Conversely, in the enclosed spectrum the black paper in the box may be called red or green at one end and blue or violet at the other. When carrying out the spectrum test it is not sufficient for the boy only to name the colours; he must point to them and indicate the beginning and end of each part.

One great advantage of Edridge-Green's system of classifying colour blindness is that it enables us to foretell what colours will be selected to match another colour when once the spectrum of that boy has been obtained. Take, for example, a boy who belongs to the two unit type. He will match all colours on one side of the spectrum provided that they are isochromatic. A light red, orange, brown and medium green would all be grouped as one colour, while, on the other side of the spectrum, violet and blue would be called by the same name; he will also confuse pure yellow with greenish-yellow, light orange and light brown. In the three unit type when red can be seen as a separate colour, the adjacent spectral colour, orange, may be

ILLUSTRATIONS TO THE ARTICLE BY DR. E. S. HORNING AND MR. K. C. RICHARDSON.

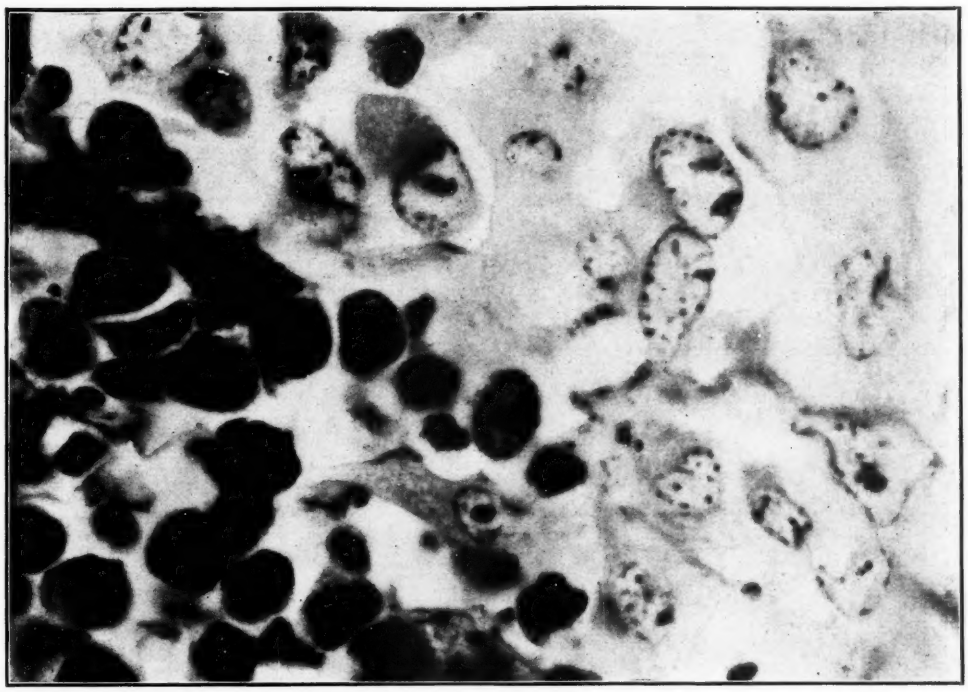

FIGURE I.

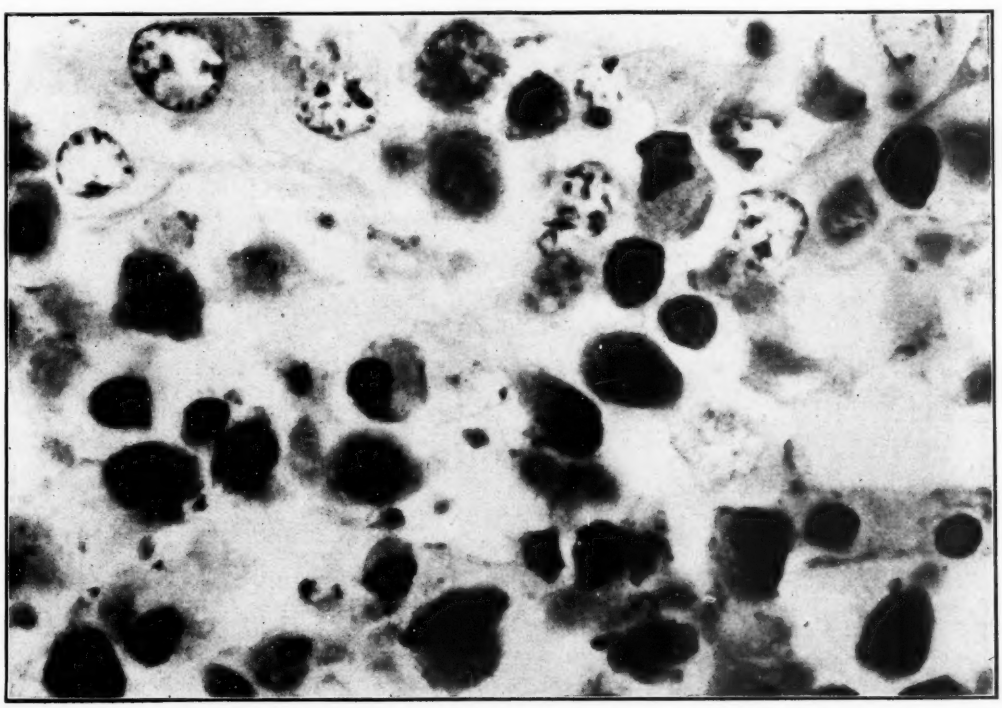

FIGURE II.

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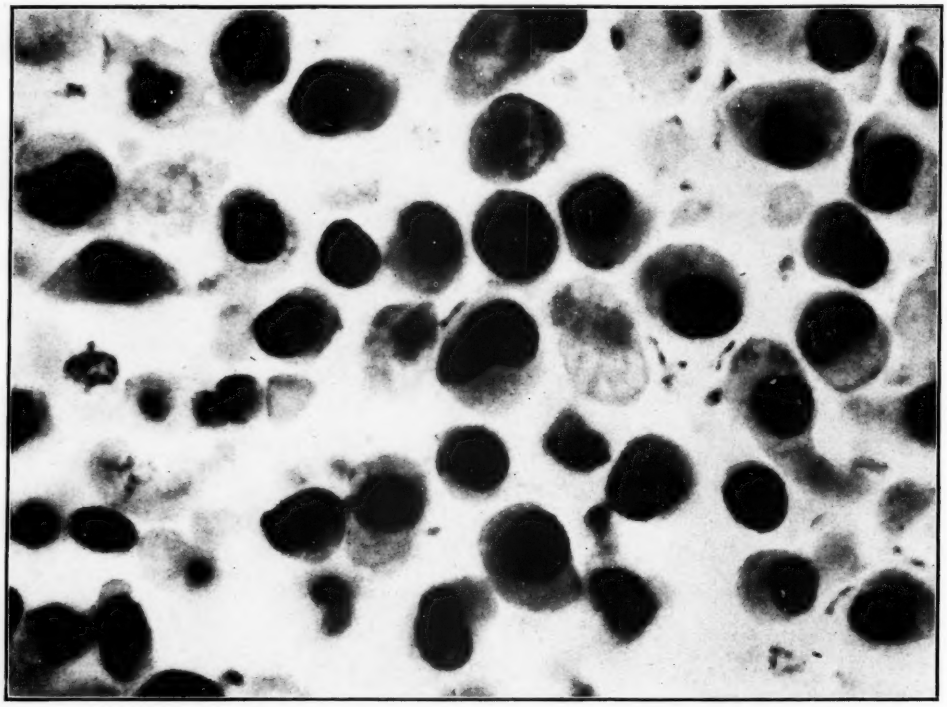

FIGURE III.

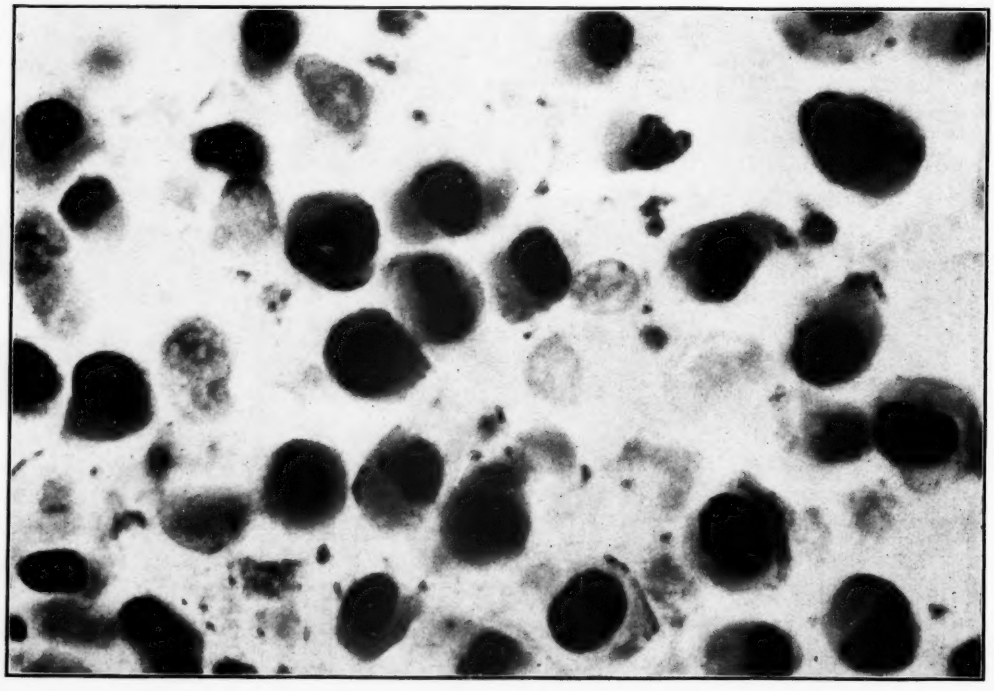

FIGURE IV.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. S. HORNING AND MR. K. C. RICHARDSON.

FIGURE V.

FIGURE VI.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. S. HORNING AND MR. K. C. RICHARDSON.

FIGURE VII.

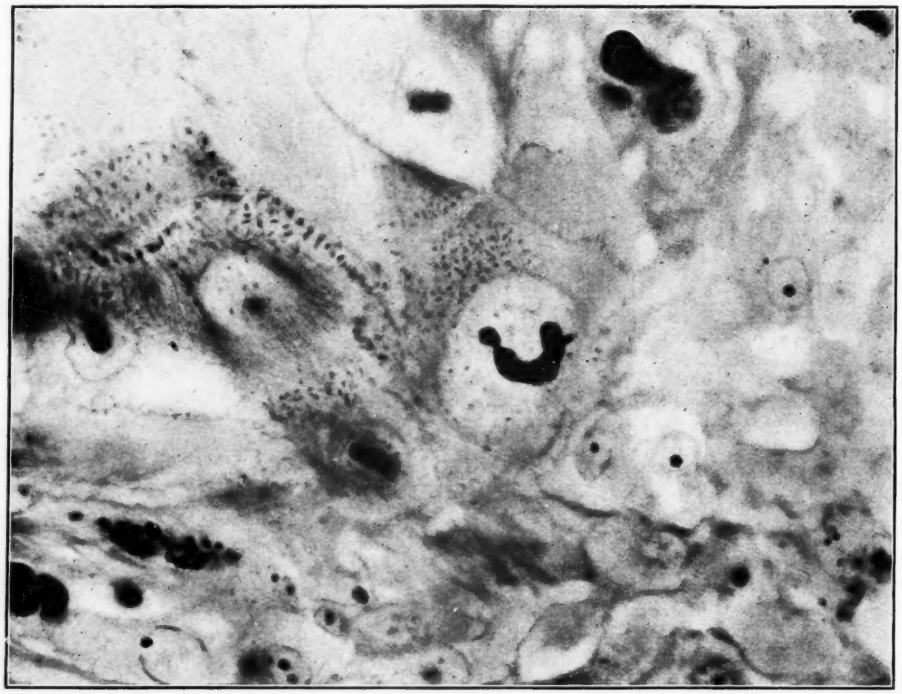

FIGURE VIII.

matched with red, provided it is a dark orange; if light orange, it will be included among the yellows. In this group green is never confused with red, for the red can be distinctly seen; it forms a separate part of the spectrum and differs from the red seen by a normal person only by extending further along the spectrum so as to include part of the orange. A similar process of reasoning will show what colours are likely to be chosen by any boy whose spectrum is known.

The results of a defect in colour vision can be seen by giving the subject a box of pastels and some coloured objects which he is required to draw. I formerly made use of flowers, chiefly sweet peas and antirrhinums, as they have many different colours and can be had during most of the school year. The best results were obtained by using pink or very dark red flowers. The pink ones are nearly always drawn as blue, since the amount of red in the flower is not sufficiently strong to be visible and the residual colour, blue, is therefore the one depicted. Where a very light pink flower is used, the result is a white or grey sketch. If the red in a dark red antirrhinum, such as the Crimson King, is not visible, the result is black or very dark blue. Mistakes are, of course, made with the colour of the leaves and stems as well. Given a sufficient number of coloured objects any colour blind boy will make mistakes. With a restricted number he may by chance select the correct colour. As a rule I made a rough sketch of the flower and of one or two leaves and the stem and asked the boys to fill in the colours, mixing them in any way desired so as to get a colour which would closely resemble the object. The results are interesting and comprise pink roses with pink leaves; a kookaburra in black and white; a sea gull with green beak and green legs and hundreds of other departures from the normal. I now use a standardized sketch with colours which are likely to be confused, placed close to one another and mark each sketch with the spectrum seen by that boy. A boy who is not colour blind, can fill in the colours in about a quarter of an hour, while the colour blind sometimes require more than an hour. In order to guard against mistakes due to colour fatigue, I get some of them to start at the top and work down; others begin at the bottom and work up; some proceed from left to right and others from right to left. The results are approximately constant for each group, no matter in which direction the boy works.

Variation.

Some of the children have been tested more than once. Variation is found no matter which test is used. There is, of course, a slight variation from time to time. Where the light is bad the results are worse; for example, dark red and dark green are almost invisible to some of these boys as coloured objects on a black cloth on a cloudy day, but may be visible during sunlight. A class examination is held in the schools every Friday morning. If a child is tested immediately after the examination, while he is still excited, the results are very often better than his normal; but if there is delay for an hour or so the results are below his normal. This is probably due to the examination stimulating attention and the fatigue which follows depressing it. Consequently if a boy shows any difficulty in carrying out the tests near the close of the school day, I get him again, if possible, the following morning and obtain his normal response. Some of those who make mistakes at night are normal in the morning, showing that while they are all right when fresh, they do not possess the normal resistance to colour fatigue when exhausted. Such persons may be a danger to the community if they are engaged in an occupation in which a recognition of colours is necessary for the safety of others. A mistake made by such a person is just as likely to result in an accident as a similar mistake made by a man who is really colour blind. I do not know whether a similar result would follow physical fatigue, but there is no doubt about its occurrence during mental exhaustion. Just how far that variation will go will depend on the individual. In an extreme case a person may have two units at night and have five or six units of colour vision by the morning. The variation is generally much less than this; the determining factors being, probably, his resistance to colour fatigue, the degree of mental exhaustion and the intensity of the colour stimulus received. Another type of variation is that which is due to education. A better discrimination of different shades of colour is generally found in the high school pupils, but no amount of discrimination will turn a two unit into a three unit spectrum. You can ask him if there is any red in the spectrum and he will say: "No." You can point to red and he will not recognize it as anything more than a dark yellow. But if he really belongs to the three unit class, then he will recognize red as soon as his attention is drawn to it. Sometimes he will recognize it if he is told to go over the spectrum very carefully and to look out for any other colours. I nearly always ask them to do this. At first I did not do so. If the boy said that there were only two colours in a spectrum and marked the beginning and end of each colour, I let it go at that without asking any questions. It is probably among these persons that this type of variation will occur; but I shall not be certain for a year or two yet when larger numbers will have been reexamined.

In addition to such types of variation due to fatigue and education, there is another which is probably much more important. In this there is a definite change in the degree of colour blindness. About 25% of all persons improve, 25% get worse and the remainder are approximately stationary. Some have improved to such an extent that I no longer regard them as colour blind; others are improving but still make mistakes and some are getting worse. What the final results will be in any one case is still a matter for conjecture. It seems to suggest that there is an instability of the colour sense in some children. This may oscillate

round an average standard for that child or the change may be progressive. Further work is required to determine the nature and extent of this change, but its occurrence is beyond question. It is not a matter of age. That has nothing to do with colour blindness. The average age for all my two unit boys is 11·5 years; for three unit boys 11·5; four unit and five unit, 12 years. That is, the average age of the colour blind boys is much the same as that of the other boys whom I examine. It is not a question of intelligence, since that would fail to account for those whose colour vision becomes worse. One boy improved from two to four units and on a third examination had relapsed to two units with a grey band between the colours. One three-unit boy relapsed to two units, while another varied from three to five units and then went back to three units. One four unit boy has changed to two units and two others to three units, while five five-unit boys changed to four units and one to three units. I should, perhaps, have stated that the examinations are conducted at intervals of approximately one year. The maximum improvement between any two examinations is three units —a change from the two to the five unit class. The maximum change in the other direction is from four to two units. No one has yet developed less than two units, but several have changed from five to six units, that is, they have become normal so far as the spectrum test is concerned. The change is not restricted to the spectrum. The wool test gives approximately the same figures—one out of four improves; one out of four gets worse and the other two remain stationary. With Ishihara's numbers the percentages are different: 10% improve, 10% get worse and 80% are unchanged. The difficulty in estimating any variation with the wool test and the numbers is that slight variations may occur and have no significance. In making up the figures for the wool tests I have regarded as a definite variation only such changes as would indicate a change in the spectrum such as the addition of green to a previous selection of reds and browns to match red, while the addition of brown to a previous selection of red and reddish brown would not be regarded as a variation. Such a standard is very arbitrary, but it is the one which I have used, with variations for each of the test skeins; since I now use the spectrum as the standard test, it is no longer necessary for me to consider any variations with wool. When I am pressed for time, the wool test is omitted. It is also difficult to estimate small changes with the numbers. The typical colour blind boy will get only one number right. But some make only three or four mistakes and yet belong to the same spectral group. The difficulty is probably due to the fact that Ishihara's test depends on colour contrast. Weak contrast will lead to a mistake just as colour blindness would, so that no change in the condition of the boy would be noted unless that change was fairly considerable. That probably accounts for only 20% showing any variation by this test, while 50% change by the other tests. Since all the colour blind members of a family belong as a rule to one group, it will be interesting to note whether they all change, the extent of the change in each and the age at which these changes occur.

Another problem is that concerned with some persons who get all the colours of the spectrum right, but who make mistakes with either the wools or numbers or both. Others get the numbers and wools right, but go wrong when indicating the colours in the spectrum. These, however, are not commonly seen. I have not come across any persons with monocular colour blindness. Although I do not examine the boys for monocular colour blindness, yet each boy who makes mistakes with the number test, is now tested with a spectroscope, each eye being examined separately. I have had this instrument for about four months only and in that time have found nine boys whose colour vision differs in the two eyes.

One boy, for example, could see blue, green, red with his right eye; with his left eye he could see blue, yellow, red and was quite certain that there was no green visible. Again using his right eye, he was just as positive that there was no yellow visible. Using both eyes on an open spectrum about fifteen centimetres (six inches) long, he could see blue, green, yellow and red, the result being the sum of the colours perceived by each eye separately.

The great advantage of this spectroscope is that it can be used without the direct rays of the sun. The disadvantage is that I have to be content with the names of the colours, for there is no other means of indicating what is seen.

Some text books state that congenital colour blindness probably does not occur in females. This is not true. I do not examine many girls, but have come across a few with congenital colour blindness. Only one of those in the metropolitan area has been tested by means of the spectrum and she is typical of the two unit class, not differing from boys of this type in any respect. The percentage is very small. In one city school I tested just over 500 girls for their colour vision without finding any defects. I have recently tested 700 girls on Eyre's Peninsula with a similar result. On another occasion I found three colour blind girls among thirty. In calculating the percentage of boys who are colour blind, the test used and the standard adopted must be taken into account. With the tests which I use and the standards mentioned 6·7% of all boys have some defect in their colour vision. Edridge-Green puts those with two or three units in a separate class and he calls them the dangerously colour blind, because their defect would be a source of danger in such occupations as navigator or engine-driver. In my statistics these come to practically 4% of the total number of boys who are tested, approximately 2% being two unit and 2% 3 unit types. The girls who are colour blind probably come to less than 0·1%. Although my apparatus is rather crude, these results are much the same as those obtained by other observers using accurate instruments.

I have come across only one boy with total colour blindness.

He differs from others with total colour blindness in that colours from the different parts of the spectrum are all confused with one another: light blue, light green, light yellow, pink, white and grey form one group, while all the dark colours are grouped together, violet, red, green, dark orange, dark brown and black. When he was being tested with the full outfit of 180 test wools he selected so many to match each test skein that, in order to save time, I took a record of those which were not, instead of those which were, selected.

Some writers refer to a peculiar look in the eyes of a colour blind person. I have tried to find this difference, but after examining about 1,000 affected persons with about 1,300 examinations and reexaminations, I have not yet seen it. When these boys talk about colours, they make use of many terms which have no meaning to a person with normal colour vision, such as light white, dark white and reddish-green. A mixture of red and green will produce brown, but this is not what they mean. Light white may be pale pink or pale blue and dark white is grey, darker pink or darker blue. They have frequently told me that brown bears a closer relation to green than to red; this indicates a shortening of the red end of the spectrum; but whenever they begin to name colours, it is better to get them to select the colour which they mean, rather than to rely on the name which they use.

Efforts to Overcome the Defect.

Many attempts have been made to overcome the defect which exists in colour blindness. These have all been failures so far. I am trying the effect of getting the boys to practise drawing coloured objects at home, but have no results to report as yet. Probably it will not make any difference to the boy, but may amuse the onlookers. After one year's practice a boy showed me his pastel book. It contained a picture of a blue kangaroo with a green tail. If it does nothing else this will convince the parents that a defect really exists and they may take steps to arrange the boy's future so that the defect will exert as little drawback on his career as possible. This takes it for granted that the observer can note the defect. A case which impressed this on me occurred last year. A gentleman objected to his grandson being told that he was colour blind and told me that when he tested the boy, he was all right. I arranged for a demonstration and found that the grandfather was also colour blind and of the same type as his grandson.

Colour blindness is a great handicap in many different professions and trades. A high school pupil told me that he could not see the green colour of boron in the flame test. I have frequently been told that horses are the same colour as grass.

A high school boy said that he had seen green horses. He knew that they were not green, for other people called them brown, but they were the same colour as grass which he knew to be green.

A teacher told me that he could not see the red flag on the golf course unless he was quite close to it when the shape and movement of the cloth made it visible. It appeared to him to be the same colour as the grass surrounding it.

In the case of teachers, the drawback is apparent only in nature study and in pastel drawing.

For example, when studying for his Bachelor of Science degree at the University, a teacher found that he could never get correct results in analytical chemistry. This made it impossible for him to continue, so he changed over to the arts course which he completed quite successfully. His colour blindness made it impossible for him to distinguish between arsenic sulphide and antimony sulphide, one being bright yellow and the other orange.

Some very instructive cases are to be found in the introduction to a book on colour blindness by Dr. Mary Collins.[4] The writer of the introduction, Dr. Drever, of Edinburgh, tells of an undertaker who was asked to supply a coffin covered with black cloth. The coffin duly arrived but the cloth was dark red. Another of his stories tells of a gentleman who one day met a lady friend in the street. She was dressed in a dark green costume which he mistook for black and it was not until he noticed her look of surprise when he began to console her on her bereavement that he realized he had made another mistake. Another story tells of a physician who told some of his confrères at a medical conference that in all the years of his practice he had never come across a case of scarlet fever. Innumerable stories can be found all through the literature about colour blind persons who were not able to distinguish ripe fruit, if red, from unripe fruit, if green.

One boy, who occasionally helps his father in his fruit shop, told me that the only difference between a ripe tomato and an unripe one is that the ripe one has a soft colour. I was not able to ascertain what he meant by a soft colour beyond the fact that it had something to do with touch as well as with sight.

Another boy lost his vacation job of picking apricots, the farmer telling him that more than one-half of the fruit in his basket was not ripe.

In those trades and professions to which I have referred it is obvious that colour is important, although one is not inclined at first to regard it as such. It is quite different when we come to those in which colour plays a prominent part. Take a painter, for instance.

One colour blind boy whose father is a painter, occasionally helps by mixing the paint, but it is always necessary to put the material to be mixed before him, as he cannot make a proper selection from unlabelled packages. What the result would be if he were turned loose to decorate a house without any help can be imagined.

Edridge-Green tells the story of a man who made a painting of a brown cat; in the picture the cat was green. I heard of a salesman who some years ago was employed in a clothing warehouse in the dress material department. His duties included sending pieces of cloth to country customers to match samples which they had submitted. Complaints were frequent that he would send red when green was wanted and *vice versa* and that if the cloth was coloured, he was just as liable to make mistakes as not. He was tested, found to be colour blind and transferred to another department. Some

Government departments have for many years recognized that a good colour sense was essential in their work. Candidates who wish to become engine-drivers and navigators have, therefore, to pass an examination in colours before they can be allowed to qualify for those positions. The signal lights used in the South Australian railways are now, I believe, a bluish-green and an orange-red. These colours are much more suitable than the former green and red signals. It is possible that some of the accidents which occur at night when motor car drivers run into safety zones may be due to the use of a comparatively dim red light in such places. Where there is a shortening of the red end of the spectrum such a light would be almost invisible. It would certainly not be red.

I was once examining a boy who belonged to the two unit group and who was consequently colour blind to red. I asked him to note the colour of the lamps at safety zones and to report to me as soon as possible. He reported that they were red; he knew that they must be red as they were danger signals and danger signals are always red.

This form of reasoning it quite common. It is sometimes successful, but when this boy was told to select a good red from a heap of coloured glass he picked out a fairly dark green. I have not yet come across a colour blind boy who wished to become a jeweller, but it is only necessary to look at the assortment of colours in the window of any jeweller to picture the confusion which might easily occur. The tests conducted with Holmgren's wools are a practical guide to what one might expect where cloth of any kind is concerned. We then find blue and violet matched with one another; brown, green and red all mixed up; while some shades of pink and blue are regarded as being of the same colour. If you try to apply these stories to industry, you will get some idea of the disability which is imposed on any boy who has the misfortune to be born colour blind.

The trades and professions which are open to a colour blind boy are those in which colour discrimination is not important, such as a lawyer, musician, bricklayer, fitter, turner, moulder *et cetera*, but the more seriously colour blind boys whom I examine get a circular containing a list of trades in which their defect would be a handicap and another list in which they might be a success. If you can offer suggestions as to additions or alterations to that list, I shall be delighted to have them.

During the last few weeks I have come across two irregular spectra.

In the first of these the boy made four mistakes with the coloured numbers, yet when tested by the spectrum he could see seven distinct colours. This is the only person with heptachromic vision whom I have met. The part called indigo was described by him as a dark colour which resembled a mixture of black, dark green and dark brown. On the following day he again pointed out this seventh colour without the slightest hesitation and indicated its position exactly as before. He was tested with the wools and got all the tests right.

The other case is probably one in which psychology is concerned as well as colour blindness.

Through the small spectroscope the boy saw a spectrum consisting of blue and white fading into black. The other eye gave a similar result. I tried him several times, even asking him if he could see any other colours such as green, yellow or red and he always replied: "No." Then I showed him a spectrum projected on to a white wall and about 12·5 centimetres (five inches) long. Without the slightest hesitation he told me that he saw four colours, blue, green, yellow and red and was able to mark their boundaries correctly. On again letting him use the small spectroscope he again told me that he could see blue and white passing into black. When I told him that he was looking at the same colours as those which were shown on the wall, he hesitated for a few seconds and then told me that they were now quite distinct. He could not understand why they were not visible previously; neither can I. His wool test confirmed the spectrum test, since he possessed four units of colour vision.

Successive Contrast.

I have tried some experiments in successive contrast trying by means of negative after images to find out if colours which are not visible in the spectrum, produce the negative after images which would be seen by a person with normal colour vision. In order to get reliable answers I have chosen two types of persons. The first are three unit types who see blue, yellow and red, but no green, and the second are those four unit types who see purple, blue, yellow and red. If they received any physical stimulus from green, they should get a red after image. In no case was this obtained; the answers were mostly "white," but a few boys said "blue." Conversely, when shown red they gave blue as the after image. I have not carried out a sufficient number of experiments to make a definite statement on these results. The object of the experiment is to find out if the defect is psychological or not. If the boy receives a green stimulus, but is not able to interpret it, then he should still get a red negative after image, since he is able to interpret the resulting red stimulus. If the defect is in the retino-cerebral apparatus, then the negative after image should be such as would be produced by those other colours which have been combined with the green pigment. So far this result is the one which I generally obtain, indicating a physical, as opposed to a psychological, cause for colour blindness.

I would like to receive suggestions and criticism for future work on this subject. Two which suggest themselves to me are that as many boys as possible should receive the spectrum test whether they pass the number test or not. Only last week I let some boys who were successful with the number test, look through the spectroscope and one of them could not see yellow even when told where to look for it. He said that green passed directly into orange and with the wool test he made the typical mistakes, selecting orange and some dark yellow wools to match the orange; light yellow and light green to match the greenish yellow test skein and yellow and greenish yellow to match pure yellow. It is thus possible that some cases are being missed. Their defects may not be serious, but they would be interesting. It would also provide some information as to the standard of colour vision in the average boy.

The other branch of this work for further investigation is that connected with variation. This

is important. As a rule I try to finish in a school by Friday night, so that it is only rarely that I get an opportunity for noting the effect of a rest from study till the Monday morning. By noting the colours which a boy says are visible in the spectroscope and taking a further note of those which he says are visible after they have been suggested to him, and then comparing these results with those obtained at other examinations held, so far as possible, on the same day of the week and at the same hour, it will be possible to give more reliable statistics obtained from a much larger number of boys. Whether these results will confirm those which I have already mentioned or not, I do not know, but there is no doubt that variation does occur. It may be that changes take place at short intervals or the period may be long. Time alone will show.

Conclusion.

In preparing this address I have been conscious of the many limitations which are necessarily imposed on me by the nature of my work. All tests must be such as can be carried out by children of a school-going age; the apparatus must be readily portable and it must be capable of being used in ordinary daylight. At the same time, it must be sufficiently accurate for diagnostic purposes, not only for extreme colour blindness, but also for those whose defect is slight, for there are many transitional types. I have omitted all references to theories of colour vision. My work is practical and, except for the terminology used for the purposes of classification, it makes no difference which theory I adopt. If the theories fit in with the facts, good and well; but if they do not, then the work can go on just the same.

In conclusion I must take this opportunity of thanking Dr. Halley, the Principal Medical Officer of the Education Department, for the help which she has at all times been only too willing to give me and to the Director of Education, Mr. Adey, for permission to read this paper.

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(1) W. Edridge-Green: "Colour Blindness and Colour Perception."
(2) W. Edridge-Green: *Ibidem*, 1891, Chapter 6, page 55.
(3) R. A. Houston: "Light and Colour," 1923, page 9.
(4) Mary Collins: "Colour Blindness," 1925.

Reports of Cases.

DEATH FROM THE BITE OF A TRAPDOOR SPIDER.

By R. N. BEAZLEY, M.B., Ch.M. (Sydney),
Wahroonga, New South Wales.

MRS. B. was bitten on the left thumb by a trapdoor spider at about 8 a.m. on January 8, 1930. The dead spider was produced and later identified as *Atrax robustus*.

I saw the patient at about 8.40 a.m. Her face was red and congested, not cyanosed. The rest of the body was pale, cold and clammy. The pulse was thready and almost uncountable and the systolic blood pressure 55 millimetres of mercury. There was incessant retching of thick tenacious mucus. In about an hour the retching gave place to increasing dyspnœa with physical signs of steadily increasing pulmonary œdema, with paroxysms of laryngeal spasm recurring every ten to fifteen minutes. The face became cyanotic and the rest of the body mottled and livid (extreme peripheral stasis). There were no muscular spasms or tremors and the physical strength was considerable, being used in energetic resistance to any attempts at treatment by mouth, by rectum or hypodermically. The mind seemed clear, but the physical reactions were so violent that one could not judge of this. There was no complaint of pain and no redness or swelling of the thumb or arm. The general condition grew steadily worse up to the time of removal to hospital at midday.

The patient was packed with blankets and hot bottles and given "Pituitrin" and adrenalin from time to time and one dose of camphor in oil, all with very temporary benefit. Later atropine sulphate was given for the pulmonary œdema without apparent effect.

A tourniquet had not been applied promptly after the bite and when I arrived it was obviously useless to put one on.

The patient was a heavily built, robust looking woman of forty-six. She had had several "nervous breakdowns" in the past four years, but she had no history or signs of other disease.

The following notes of the patient's condition while she was in the Royal North Shore Hospital of Sydney, have been kindly supplied by Dr. M. J. Hudson, Assistant Medical Superintendent.

The patient was admitted at 12.10 p.m. She was restless with stertorous breathing. A moderate degree of cyanosis was present with frothing at the lips. The patient was conscious but reluctant to answer questions. About every ten to twenty minutes she had clonic contractions of arms and legs, lasting about two minutes, accompanied by laryngeal inspiratory stridor and increased cyanosis. Between the clonic spasms both upper and lower limbs were slightly spastic. The knee jerks were increased. Her eyes were staring; the pupils were small and did not react to light; the corneal reflex was sluggish. The pulse rate was rapid and almost imperceptible.

At the site of injury were two very minute punctures. On excision of these two definite thrombosed small vessels were seen. No surrounding œdema was found.

At 2 p.m. the patient was comatose, but was still having laryngeal spasms about every quarter of an hour, lasting one-quarter to one-half minute. She died suddenly at 5 p.m.

The treatment adopted was the application of a tourniquet, excision of the area surrounding the punctures and application of potassium permanganate, together with the administration of stimulants and of glucose and saline solution *per rectum*.

Post Mortem Findings.

The autopsy was performed by Dr. A. A. Palmer, to whom I am indebted for the following notes.

Autopsy was performed at 10.45 a.m. on January 13, 1930.

The body was that of a stout woman, aged forty-seven years. Fine froth was present at the lips and throughout the air passages.

There was a small wound on the front of the left thumb, stained with permanganate of potash. Beneath this there was no thrombosis in the vein and no extravasation of blood into the tissues. The lungs were œdematous, red and fleshy looking on section. The right lung was adherent to the chest wall and the left had a few petechiæ beneath the pleura.

The heart was flabby and fatty infiltration was present. There was some atheroma of the mitral valve and of the aorta. The blood was very dark. There were some clots in the right chambers. Congested areas were found in the mucous membrane of the stomach towards the cardiac end. The liver was of a pale yellowish colour in places, congested in others. The kidneys were congested. The brain was congested and there were a few atheromatous patches in the arteries.

Microscopical examination was carried out by Dr. E. L. Morgan, Principal Microbiologist, of the Department of Public Health, who has supplied the following report.

Sections of lung were extremely congested and œdematous distension of many of the alveoli was found.

Sections of cerebral cortex manifested no definite abnormality.

Sections of liver were congested and a slight degree of fatty degeneration of the liver cells was present.

Sections of kidney manifested a patchy congestion of the cortex and medulla with degeneration of the epithelium of the convoluted tubules, possibly a *post mortem* change. The glomeruli were not affected.

In sections of the congested patch in the stomach wall dilated blood vessels could be seen extending throughout the submucosa.

Reviews.

OPHTHALMOLOGY.

"THE EYE IN GENERAL MEDICINE," by Dr. Maitland Ramsay, is one of the most important books on ophthalmology published in the last ten years.[1] In a primitive form it appeared twenty years ago under the title "Diathesis and Ocular Diseases." Since then the theme of the book has expanded as a result of the continued careful observation and mature consideration of its gifted author. Recently Dr. Maitland Ramsay's work at the Saint Andrew's Clinical Institute has given him unique opportunity to substantiate his statements.

To him, as it should be to us all, the eye is one of Nature's laboratories open for inspection. It is easily accessible; its media are transparent and it offers a highly magnified view of its posterior tissues. The observer sees the column of blood contained within arteries which are terminal branches of the cerebral arteries. The vessel walls are transparent and any loss of this quality indicates degeneration. The venous blood can be seen as it courses on its way to pour into the cavernous sinus. The retina may be considered as the extracranial portion of the cerebral cortex and the stalk of the nerve as homologous with the association fibres in the brain. Krogh has demonstrated the vital rôle played by the capillaries and their ability to respond to the hunger of the tissues. In the fundus with yellow-green light capillaries, identical with those elsewhere, can be clearly seen. The value of this is emphasized by the author's words: "The capillaries are the most active, purposive and dynamic part of the vascular system and all vital processes take place through their walls." The capillaries and the essential tissue in any organ, whether it be muscle fibre in the heart, glomerulus in the kidney or rods and cones in the eye, are the "organ in miniature" and their interreaction determines functional activity. "There is no more convenient field for the direct observation of vital process either physiological or pathological than the eye." The transparency of ocular tissues adds to the ease with which alterations in them, due to disease, can be detected against the red background of the fundus. Not only are the refractive media transparent, but also the retina and the walls of the retinal vessels. In the latter the trained observer can watch the early and late changes in arteriosclerosis, diabetes, nephritis and leuchæmia. These must be identical with the unseen alterations in other organs. Patients suffering from arteriosclerotic retinitis usually die from failure of the vascular system. Many succumb to cerebral hæmorrhage; of those who escape apoplexy, a great number dies of heart failure, notwithstanding the fact that albumin has never appeared in the urine throughout the whole course of the illness.

Dr. Maitland Ramsay draws interesting parallels between the ocular lesions produced by the pneumococcus and those due to it in the lungs; the pain due to the spasmodic contraction of unstriped muscle, whether it occurs as *angina pectoris*, strangury, colic or photophobia; and between hippus and a form of cardiac irregularity. By such instances he developes the idea of "the unification of vital processes." "Some in recent years have tried to promulgate the idea that spectacles concern the optical part of the eye only and have nothing to do with the disease. That is most pernicious doctrine, because in no circumstances can the living eye be treated simply as an optical instrument. It is not only in the body; it is also of the body; and the proper treatment of its optical defects requires the physician's skill just as much as does the treatment of its diseases. It must never be forgotten that in the eye, just as in any other part of the body, the limits of muscular capacity must be respected and the amount of work regulated accordingly."

The practitioner still exists who is annoyed when the chemist supplies a cold cure for every cough and a pill for every abdominal pain and yet who sends his eye-strain patients to those qualified only to manufacture spectacles; he would learn much of value to his patients by a perusal of the chapter entitled "The Medical Aspects of Eye Strain." Two points require reemphasis: "It is the constant effort to compensate a low error of refraction that causes the distress. It is the overwrought muscle that goes into spasm and cramp and the results are similar in whatever part of the body unstriped muscle is found." In the majority of these persons there is little or no loss of visual acuity. The second point is expressed by Dr. Ramsay as follows: "It is important to remember that of all the unstriped muscles of the body it is only the ciliary muscle and the cardiac muscle that can be forced by an effort of the will to continue working after Nature has called out for rest." The danger here is great.

[1] "The Eye in General Medicine: The Constitutional Factor in the Causation of Disease with Special Reference to the Treatment of Diseases of the Eye," by A. Maitland Ramsay, M.D., LL.D.; Second Edition of "Diathesis and Ocular Diseases"; 1929. London: Baillière, Tindall and Cox. Demy 8vo., pp 265. Price: 12s. 6d. net.

AUSTRALIAN SNAKES.

A DESCRIPTIVE work on the snakes of Australia has long been needed. Krefft's classical work is sixty years old and Waite's book was published thirty years ago. Mr. Kinghorn has fulfilled this need by producing an excellent pocket book with brief but lucid descriptions of thirty-four non-venomous and one hundred and five venomous species.[1] In the introduction the author in addition to giving a good deal of general information about snakes, deals delightfully with some of the common fallacies concerning them.

For the purpose for which the work is intended, namely to provide persons interested in snakes with sufficient information to enable them to recognize the numerous species, the book is wholly admirable and the simple keys which Mr. Kinghorn has made are in nearly all cases sufficient for identification. The coloured plates illustrate well the salient features of the species.

Mr. Kinghorn is to be warmly congratulated upon having produced this very useful little book and the publishers also for the care and attention that they have devoted to its preparation, notably in the matter of the illustrations.

It is a little unfortunate that Figure VI should illustrate two faulty positions for the application of ligature, the forearm and the leg, where, owing to failure to obstruct interosseous vessels ligature would be ineffective.

It is to be hoped that the success of this work will encourage Mr. Kinghorn to extend its scope and in a larger volume to provide fuller descriptions with synonyms and literature for the benefit of those who are working in this field.

[1] "Snakes of Australia," by J. R. Kinghorn, C.M.Z.S., with a foreword by Heber A. Longman, F.L.S.; 1929. Sydney: Angus and Robertson, Limited. Imp. 32mo., pp. 198, with illustrations. Price: 10s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 22, 1930.

Poliomyelitis.

DURING the past five years we have reminded our readers from time to time of the movement in Melbourne and Victoria to check the ravages of acute poliomyelitis. The aid of the Commonwealth Serum Laboratories was obtained and a substantial supply of the serum of persons convalescent from poliomyelitis or of those who had had poliomyelitis within two or three years, was prepared under aseptic precautions ready for use. Dr. Jean Macnamara took charge of the campaign and lent her aid to any medical practitioner who sought it, in establishing the diagnosis of poliomyelitis before the onset of paralysis. The success of her work has been recognized by the whole medical profession. The disease appears both in epidemic form and sporadically and the extent of the individual outbreaks varies a great deal. Up to the present the result of the campaign has been to reduce mortality and to lessen the incidence and severity of the paralysis.

For a considerable time Sir Charles Clubbe has been endeavouring to awaken the interest of the public of New South Wales in the same problem. He has had a survey made at the Royal Alexandra Hospital for Children of all patients who have been treated for poliomyelitis, partly to ascertain the extent of the disease and partly to determine the frequency of residual paralysis. He realized that a campaign similar to that in existence in Victoria, would be costly and would depend for its success on the skill of the practitioner called upon to lead the attack. It was suggested that an invitation should be sent to Dr. Jean Macnamara to initiate a campaign in Sydney and to train someone to continue it when she returned to Melbourne. Owing to the lack of interest on the part of the public no progress was made in the early part of 1929. Later an appeal was made through the daily press and the response was immediate and generous. It was then determined to appoint a committee, to be known as the Infantile Paralysis Committee. Sir Charles Clubbe is the Chairman, Dr. R. Dick, Director-General of Public Health of New South Wales, Dr. E. W. Fairfax, Dr. E. H. M. Stephen and Dr. A. H. Tebbutt are members. It has been decided to appoint Dr. Karen Helms the medical officer. She will study the methods employed by Dr. Macnamara in Melbourne and will then apply these methods in Sydney. The serum of persons who have suffered from poliomyelitis, will be obtained and will be prepared for use by Dr. A. H. Tebbutt at the Royal Prince Alfred Hospital. A circular memorandum has been drawn up by the committee and sent to every medical practitioner on the roll of the New South Wales Branch of the British Medical Association. It is hoped that the medical profession in the State will respond promptly and willingly to the invitation to avail itself of the expert assistance provided by the committee. The aim in a few words is to diagnose poliomyelitis as soon as it occurs in every affected child, so that serum may be injected and paralysis prevented. The symptoms in the initial stage are somewhat vague, but an experienced clinician is capable of deciding within a short time whether or not any febrile attack is poliomyelitis. The symptoms are fever, headache, irritability and drowsiness, pain in the neck, at times vomiting, tremor of the hands, localized sweating, photophobia and retention of urine. All this has been admirably described by Dr. Macnamara in her articles that have appeared in this journal. The objective evidence of the disease is obtained from the so-called spine sign. She describes this as a voluntary disinclination to flex the spine anteriorly because it hurts. She has also recorded her experience of the diagnostic value of examination of the cerebrospinal fluid. She emphasizes the necessity of being prepared to inject convalescent serum if lumbar puncture is performed. It will be obvious to all general practitioners that the proffered help of an experienced medical officer represents a great advantage to all concerned. They will be wise to suspect acute poliomyelitis whenever they encounter

a febrile condition in a child that cannot be diagnosed as morbilli or any of the other common infections. If poliomyelitis cannot be excluded, the assistance of Dr. Helms should be sought. In Melbourne as elsewhere the mortality among children treated with serum in the preparalytic stage has been reduced to a minimum. The death rate among children not treated with serum has been as high as 21·6%. To achieve the low mortality, it is essential that the little patients should be treated at a time when the diagnosis is difficult. Not only will many lives be saved, but the residue of paralysis will also be greatly reduced. The severe forms of paralysis can be prevented altogether when serum is administered at an early date.

A word should be devoted to the question of the prophylaxis of poliomyelitis. It appears that the incubation period of the disease is usually about four days. The general practitioner can perform a very valuable service if he assists the Department of Public Health in an endeavour to trace the source of infection of each child in whom the diagnosis can be made in the preparalytic stage. It is assumed that the virus gains an entrance through the naso-pharynx. Whether the virus is still to be found in the naso-pharynx after recovery from an attack is not known. It is impossible to speak of the carrier state, since the virus cannot be demonstrated directly. Although there is evidence that the immunity conferred by an attack lasts for a considerable time and that the immune bodies can be demonstrated in the serum years after the attack, the virus may lie dormant and inactive somewhere in the convalescent's body, ready to acquire renewed virulence as soon as it is conveyed to a susceptible child. It is probable that in epidemics the disease is spread from sufferer to susceptible child more frequently than from a carrier. If this proves to be so, it will be essential in all campaigns aiming at the eradication of the disease to search for the sources of infection among those children who have had the disease either in a fully developed form or in a mild, unrecognized and short-lived fever. In Victoria and New South Wales the general practitioner has a splendid opportunity of helping to gain a complete mastery over a disease that still plays havoc with young lives.

Current Comment.

MENTAL DEFICIENCY IN AUSTRALIA.

THERE have been several attempts at estimating the incidence of mental deficiency in Australia. Investigations have been carried out by educational and other public authorities in different States and a committee reported on mental deficiency to the tenth session of the Australasian Medical Congress in Auckland in 1914, but the returns represented only a portion of Australia, so that no general statistics were presented. Last year the Inspector-General of Mental Hospitals of Victoria, Dr. W. Ernest Jones, at the request of the Federal Health Council, carried out an investigation, the report of which has just been issued.[1] The survey was made with the assistance of the Department of Education and the Lunacy Department of Victoria and of other bodies with defectives under their care. The figures, therefore, deal with defectives throughout the Commonwealth who are known to the education authorities, and also those who are inmates of institutions. While the estimates of the more severe grades of mental defect, idiocy and imbecility and of those with severe physical disease are likely to be fairly complete, a considerable number of the feeble-minded and moron types, especially those above school age, must have eluded observation. Dr. Ernest Jones estimates that not less than 10% of invalid pensioners, although their mental state is not recognized, belong essentially to the category of mental defectives. No doubt most of these individuals are sufficiently well cared for in their homes and as pensioners are a smaller charge to the community than they would be if admitted to institutions, but there is no supervision in order that pensions may be granted and used to the best advantage. The returns contain the following figures of defectives of school age on the registers of schools and in mental and other institutions: idiots 0·08%, definite mental deficients 0·69%, borderline 1·35%, all classes 2·12%.

The proportion of defectives in country districts has been found to be nearly double that in the cities, while urban figures are intermediate. Dr. Ernest Jones gives 2·89% of defectives among children of school age as his general estimate for Australia. This means that 22,217 persons between the ages of six and fourteen years are mentally deficient.

In a detailed survey[2] carried out during the past three years in six representative areas, industrial, urban and rural, in England and Wales, each with a total population of about 100,000, it was ascertained that the various grades of mental defect in children of school age were: idiots 0·2%, imbeciles 0·78%, feeble-minded 5·02% and all grades 6·0%.

The English survey was carried out by a medical man, Dr. E. O. Lewis, with psychological training

[1] "Report on Mental Deficiency in the Commonwealth of Australia," by W. Ernest Jones, Department of Health of Australia, 1929.

[2] Report of the Mental Deficiency Committee, England, 1927.

and educational experience, who investigated the children in person and also by visits to their families came across many other defectives who would otherwise have been overlooked. The returns in the Australian survey were for the most part made by the head masters of schools. In New South Wales alone the diagnosis was made by the medical officers of the Department of Education and the returns are strikingly different from those from other States. Thus the personal equation renders strict comparison impossible.

In regard to the provision for mental defectives, the helpless and antisocial are dealt with under existing laws in mental institutions, although accommodation in some parts is neither adequate nor up to date. On the other hand, little is being attempted for children with the milder types who can be trained to be self-supporting and law-abiding, but who, if neglected either at home or at school, tend to grow up into dependency and crime. The Education Department of New South Wales has established a residential school for trainable defectives at Glenfield which is a model of its kind, but which of necessity cannot accommodate a tithe of all those children who might benefit there. Glenfield perhaps serves a more important function of providing experience for teachers who will later be in charge of special classes in the day schools. Dr. Ernest Jones remarks that in general in Australia the provision of special schools and classes is quite inadequate. The following extracts from the English report are of interest in this connexion.

> Our experience in the course of the present investigation confirms us in the view shared by many workers in this field, that it is the lack of proper training and the failure to secure proper occupation which gives rise to the antisocial behaviour of large numbers of the higher grade defectives. . . . For this group of defectives the most potent socialising factors are education of a practical nature during youth and adolescence and constant employment later. . . . The results already achieved by the practical training given in many of the day special schools of our large towns prove that much can be done to ensure that the stable feeble-minded children become useful members of the community.

Thus constructive work during the all-important years of childhood is the responsibility of the education authorities, while the lunacy departments must provide for those who need segregation in early years, and for those who, having passed the school age, on account of the fixity of their habits are less amenable to socializing and educational influences. The multiplication of special classes in day schools, with better provision for practical training, would be of real benefit and much less costly than the building and maintenance of institutions.

Mental deficiency should not be dissociated from the wider problem of conduct disorders in childhood. While fundamentally this is a biological problem concerning heredity, growth, nutrition, infection, trauma and so forth, yet the treatment of the patient often involves educational and other adjustments which do not fall within the province of general medicine. For this reason there have been established in many countries, notably in the United States of America, in the United Kingdom and here in Melbourne under the direction of Professor R. J. A. Berry, child guidance clinics where an all-round study of behaviour problems in childhood is carried out by a team of workers. The medical director both deals with the physical aspect and after consideration of reports from his colleagues and from his own observation, prescribes the appropriate treatment. A psychologist, usually a layman, preferably with educational experience, carries out examinations on the intelligence and the manual and practical abilities of the child, recommends the education and vocation for which the child seems best fitted, and keeps the clinic in touch with the schools, technical colleges and establishments for occupational training. The third member of the team is the social worker whose sphere of activity includes the investigation of the social environment of the child, especially of the home conditions. She arranges admission to institutions, colonies, boarding-out homes, acts as a probation officer and supervises the carrying out of the director's advice in the home where the behaviour problem arises out of domestic discord or other situational difficulty. The child is handled in such a clinic in the only scientific way, as a psychobiological problem with medical, educational and sociological aspects. Both Professor Berry[1] in the report on his own work and Dr. Ernest Jones have put forward a plea which cannot be overlooked, that the example of other countries should be more widely followed in Australia. Much is already being done for the physical welfare of youth, but purely medical or surgical treatment falls far short of an ideal. While it cannot be claimed that any considerable proportion of the insanities may be traced back to modifiable mental or physical influences operating in childhood, it must be admitted that a good deal of neurosis, dependency and delinquency is preventible. However great the bias of heredity and constitution, the post-natal environment is still of importance and there is a reasonable hope that the correction of faulty training and attitudes as well as of physical handicaps in childhood may be of value in the prevention of nervous breakdown at a later age. Childhood has been termed the "golden period" for mental hygiene. The medical profession has a responsibility in this matter.

THE WALTER AND ELIZA HALL INSTITUTE.

THE tenth annual report of the Walter and Eliza Hall Institute of Research in Pathology and Medicine reveals increased activity and a greater number of published papers than last year. The Commonwealth Department of Health has contributed the sum of £4,000 to enable the director and his staff to carry out researches on Australian snake venoms, on staphylococcal and streptococcal infections, on hydatid disease and on poliomyelitis. Many of the papers were published in our columns. The work is of a high order.

[1] Report to the Edward Wilson (*The Argus*) Trust on Mental Deficiency in the State of Victoria, by R. J. A. Berry, 1929.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Hypersensitiveness to Arsphenamine in Guinea-Pigs.

N. B. SULTZBERGER (*Archives of Dermatology and Syphilology*, November, 1929) states that the possibilities of sensitizing the skin in guinea-pigs by means of a single intradermal injection of a small quantity of "Neoarsphenamine" offer a unique field for experimental research on the subject of skin hypersensitivity. It is ideal because the test object is the guinea-pig, the method of sensitization is simple and the substance employed is one which plays an important rôle in hypersensitiveness in human beings. In his observations the author used the technique adopted by Frei; he describes this in detail. The first questions investigated were those of prophylaxis, that is the prevention of sensitization and desensitization. The author holds that the application of knowledge on these subjects in the treatment of various skin diseases seems to have made little advance, whilst the practical value of desensitization and prevention of sensitization in allergic asthma and hay fever has been well established. Moreover, it is true that certain successful desensitizations, both specific and non-specific, have been reported in eczemas, occupational dermatitides and arsphenamine dermatitis, yet it is the exception rather than the rule. In the first series of experiments twenty-three guinea-pigs were prepared by an intradermal injection of "Neoarsphenamine." Twenty-four hours after the intradermal injection six of the animals received one-third of the minimal lethal dose of "Neoarsphenamine" intracardially. A skin hypersensitivity to "Neoarsphenamine" could be observed in sixteen of the seventeen animals that received no intracardial injection. This hypersensitivity showed itself in twelve animals by a flaring up of the area of injection from six to fifteen days later. This was confirmed in sixteen hypersensitive animals by a second intradermal injection of "Neoarsphenamine." Those animals which received an intracardial injection, at no time showed signs of hypersensitivity by either a flare-up or a reaction of hypersensitivity to the intradermal test after thirty-one days. A second intradermal test was applied to the entire series seventy-eight days after the first injection and the only animals that still showed protection against sensitivity, were those intracardially treated. The experiments suggest that the hypersensitivity increases in strength after the sixth day. It was noted that the reaction of hypersensitivity differed from the flare-up in its shorter incubation and course as well as in the greater severity of its inflammatory and destructive processes. The author calls attention to the certain outward similarities between these phenomena and another form of sensitization, that is, anaphylaxis. The fact that prevention of sensitization to arsphenamine was obtained by injection of arsphenamine does not represent proof of specificity. It is also not known, when the effect of the intracardial injection is applied, whether the entire process is cellular, humoral or both. It is concluded, therefore, that the sensitization of guinea-pigs to arsphenamine is an allergic phenomenon in the sense of Pirquet's original definition. The occurrence of this allergic condition can be prevented by an intracardial injection of the allergen twenty-four hours after the potentially sensitizing intradermal injection and lasting more than seventy-eight days. In view of the fact that these experiments could not be regarded strictly as a desensitization, a further series of experiments was performed and animals rendered hypersensitive previously in the above experiments, were utilized. An attempt was made to desensitize twelve sensitized animals by intracardial injections fourteen days after intradermal preparation with arsphenamine. Six of the animals received an intracardial injection of arsphenamine identical with that used to prevent sensitization and the other six animals received an equivalent intracardial injection of gold sodium thiosulphate. Neither of these measures was effective in desensitizing the guinea-pigs and they reacted to subsequent intradermal tests with arsphenamine fully as strongly as the sensitized, but not intracardially treated controls. The intracardial injection of arsphenamine caused no anaphylactic shock, even in the hypersensitive animals.

Toxic Reactions Produced by Trinitrophenol (Picric Acid).

C. C. DENNIE, W. L. MCBRIDE AND P. E. DAVIS (*Archives of Dermatology and Syphilology*, November, 1929) record the results of their experiments and observations on the effects of the use of picric acid in surgical practice and as a base in certain ointments for application in skin conditions. They state that about 4% of persons are naturally sensitive to the effect of picric acid. Trinitrophenol, used externally in any form, is likely to be followed by disagreeable or dangerous results in a certain percentage of subjects. They recommend the use of tannic acid to sterilize the field of operation or for use as a base for ointments.

Scleroderma.

J. E. M. WIGLEY (*Proceedings of the Royal Society of Medicine*, December, 1929) reports the occurrence of scleroderma in a boy, aged seven and a half years. He had been knocked down by a bicycle four years previously and his nose had been bruised and swollen. His hair had become grey in patches two years previously and hard patches had appeared on the skin of the scalp and face one year and a half previously. There were thickened ivory coloured areas of skin on the left cheek and the side of the nose and this condition extended under the chin. The left *ala nasi* was drawn upwards and the whole of the side of the face had an atrophic appearance. There were two similar patches in the scalp. No other abnormality was present and the blood serum had failed to react to the Wassermann test. In the discussion it was pointed out that scleroderma is rare at this time of life, though it has been described in infants. F. Parkes Weber said that scleroderma of this type was seen chiefly in young people and seemed to affect the skin and the subcutaneous tissues. He divided localized scleroderma into three groups. The first was a superficial form, characterized by glossy patches, the skin only being involved. The second group was one in which both skin and subcutaneous tissue were involved, as in the patient shown. The third group comprised rare examples, apt to be wrongly diagnosed, in which there were patches of atrophy of the subcutaneous fat without any real involvement of the skin; the term scleroderma was really a misnomer for this group.

Acquired Ichthyosis.

E. G. GRAHAM LITTLE (*Proceedings of the Royal Society of Medicine*, October, 1929) reports the occurrence of acquired ichthyosis in a woman, aged twenty-four years, who had been working in a radium factory six years previously. After the skin of her fingers had begun to peel, she was transferred to another part of the factory. She subsequently suffered from boils and was treated for this condition with arsenic. The arsenic was given in two courses. In the interval between the courses the ichthyotic manifestations appeared. Nearly the whole body was subsequently involved. In the discussion it was held that the radium had not been responsible, since the fingers were not involved in the ichthyotic changes. H. C. Semon thought that the condition was seborrhœic in nature and A. M. H. Gray regarded it as an arsenical dermatitis.

Sugar Metabolism in Acne Vulgaris.

A. STRICKLER AND M. A. SAYLER (*Archives of Dermatology and Syphilology*, November, 1929) have performed a series of experiments, the results of which have been tabulated, to investigate further the relationship of sugar metabolism to *acne vulgaris*. The study was undertaken to determine whether or not hyperglycæmia is limited to a particular type of acne; whether the increase in the blood sugar is constant throughout the disease and whether the blood sugar is influenced by the limitation of sugar and starchy food diet. The series comprised thirty-nine patients. The first determination of blood sugar concentration showed that twenty-eight of them were normal, two had hyperglycæmia and nine had definite hypoglycæmia. Of those on a restricted carbohydrate diet, a greater number had an increase in their blood sugar than a decrease.

UROLOGY.

Traumatic Rupture of the Urethra.

W. I. DE C. WHEELER (*Irish Journal of Medical Science*, June, 1929) recounts his experiences in six cases of complete rupture of the urethra. The accident is comparatively rare and on account of the general lack of experience, treatment is not standardized and varies a good deal. The rupture is nearly always in the bulbous portion of the urethra, but in one of the six cases under review it was just anterior to the neck of the bladder. The author states that the first thing to do in all urethral ruptures is to perform suprapubic cystotomy; this gives the surgeon at once the advantage of examining the bladder neck and the prevesical space and deviating the urinary stream and finally of performing retrograde catheterization to identify the proximal end of the urethra should he decide to proceed at once to a perineal operation. All contused tissue and blood clot are removed from the perineum and the trimmed ends of the urethra are sutured by interrupted stitches over an inlying rubber catheter. The author believes in retaining this catheter as well as a suprapubic drain from at least two weeks and describes a special instrument for performing the retrograde catheterization and drawing the rubber catheter into the bladder. The bladder end of the catheter is attached to the rubber suprapubic drain by means of a thread and this thread serves to pull the catheter out through the suprapubic tube at the end of four days, so that a fresh catheter attached in its wake may be left inlying for another four days and so on. The perineal wound is closed up completely. By this original method the author has secured good results with a minimum of discomfort and complications during the convalescence.

Movable Kidney.

B. A. THOMAS (*Journal of Urology*, December, 1929) publishes the results of his study of nephroptosis and makes a plea for the operation of nephropexy, when modern diagnostic methods definitely give the indication for treatment. He states that practically the whole medical profession is still under the sway of teaching resulting from the reaction led by Glémand against the over-radicalism of Edebohls and his followers. The operation fell into discredit because kidneys were fixed indiscriminately at one period. Moreover, the technique of the operation was often faulty and the proper immediate and remote after care was not carried out. It is undoubtedly a fact that at present under the influence of the adverse teaching in medical schools many patients are condemned to spend the rest of their lives in more or less continual, sometimes disabling pain. In diagnosis all points, general and local, should be considered. The question of any neurotic element or of associated general visceroptosis should be solved and operation should never be advised in such circumstances without a preliminary trial of the effect of one to two months' rest in bed with a fattening diet and then the application of a proper abdominal support. In diagnosis pyelograms and ureterograms should always be added to the routine urological examination and they should be made in the erect as well as the supine position. Palliative treatment by belts *et cetera* is contraindicated when severe infection, calculus, tumour or other complication exists, when the degree of kinking, ptosis, torsion *et cetera* disclosed by a pyelogram is of more than a medium grade of severity, when residence in a remote district or country is obligatory or if the patient is a manual or heavy worker. Very many variations of the nephropexy operation have been described, but the essential points are thorough mobilization of the kidney and upper part of the ureter and the separation of all adhesions, bands and fat. The method of Kelly, the employment of three triple mattress sutures, is recommended and at least one of these sutures passes up through the muscles of the eleventh intercostal space. At the end of the operation the kidney is well behind the twelfth rib. The patient should be kept in bed for at least four weeks.

Stricture of the Female External Urinary Meatus.

M. L. BOYD (*Journal of the American Medical Association*, June 29, 1929) calls attention to the fact that congenital narrowing of the external meatus of the female urethra, long disregarded by members of the medical profession, actually does exist and is often the cause of infective troubles in the urinary tract. Occasionally strictures in this situation are acquired, but, however formed, they have a uniformly bad effect. Such a narrowing is a not infrequent cause of so-called pyelitis in little girls and of urinary infection whether of the bladder or kidneys in older females. If the narrowing is noticed, it is as a rule dilated only to allow a cystoscope to enter, whereas the proper treatment is meatotomy as would be done in the male. The meatotomy incision is placed on the floor of the outlet, as in the male, and its effect in allowing a freer outflow of urine is often the deciding factor in the cure of a urinary tract infection.

Pneumo-Pyelography in Tumours of the Renal Pelvis.

J. BEDRNA AND J. SIMON (*Journal d'Urologie Médicale et Chirurgicale*, May, 1929) consider that in addition to its other decided advantages pneumo-pyelography has rendered possible the exact diagnosis of tumours of the renal pelvis including small ones. They present some sodium iodide pyelograms which give no hint of the presence of a neoplasm, and an air pyelogram taken from the same patient shows up the tumour quite clearly by contrast with the air. Even small papillomata which are only very slightly radio-opaque, will show up by contrast when surrounded by air. With large pelvic tumours a filling defect occurs with sodium iodide pyelograms, but the diagnosis is even more clearly and certainly obvious when air is used. Pneumo-pyelography is without danger when properly employed. The size of the ureteric catheter used should not be greater than number 5 French. The insufflation of air is made with a twenty cubic centimetre syringe and should be made gently and slowly. Among three hundred and forty-five patients in their clinic the authors have observed no after troubles at all.

Intravenous Pyelography.

T. HEYNTSCHAK (*Zeitschrift für Urologie*, October, 1929) attacks the difficult problem of attempting to delineate radiologically the renal parenchyma, as well as the pelvis and calyces by means of radio-opaque chemicals introduced into the body by intravenous injection. Sodium iodide is the best chemical for this purpose, as it fulfils the necessary requirements, namely, that it undergoes no change in the body, is not toxic even in fairly high doses and possesses a high degree of solubility in water, the latter requirement arising from the need of introducing a concentrated solution into the blood so that it will be concentrated in the urine. The author has injected thirty to sixty cubic centimetres of a 20% sodium iodide solution, that is, six to twelve grammes of iodide. Although useful nephro-pyelograms were obtained only in a small proportion of all cases, the author is optimistic about future progress along these lines. To help matters he proposes that "Pituitrin" should be given to increase the concentrating power of the kidneys and atropine to depress pelvi-ureteral peristaltic action.

The Colliculus Seminalis in Urinary and Sexual Disorders.

R. C. BEGG (*British Journal of Urology*, September, 1929) considers that pathological changes in the *colliculus seminalis* and the posterior part of the urethra are frequent causes of sexual and urinary disturbances. Three case reports are published to illustrate respectively retention of urine, persistent urethral discharge and sexual irritation produced entirely by lesions of the *colliculus*. As a rule the treatment is to destroy such lesions by the high frequency current. The author considers also the mechanics of ejaculation and thinks that in erection the *colliculus* is thrust forward by the accompanying congestion while the ejaculatory ducts are straightened out. The stream of semen is thus directed forwards towards the anterior part of the urethra where the cavity of the bulb acts as a distributing reservoir. The rôle of the *colliculus* in the sexual life of the individual has been exaggerated, as it contains no erectile tissue and no special nerve endings.

Special Abstract.

MATERNAL AND CHILD WELFARE IN AUSTRALIA.

DAME JANET M. CAMPBELL, Senior Medical Officer for Maternity and Child Welfare, of the Ministry of Health of England, has been engaged for some time in an investigation into the mortality and morbidity of Australian mothers and their infants and has issued her report. In view of the importance of her findings we publish a full summary.

Maternal Mortality.

A comparison is made between the maternal mortality rates in the several States of Australia and those in England and Wales. The figures are given of deaths per thousand live births during the eighteen years from 1911 to 1928. In New South Wales the rate in 1911 was 5·2 and in 1928 it was 5·95. In the intervening years it diminished to 4·25 in 1921 and rose to as high as 6·54 in 1927. The average for the period is 5·34. In Victoria the rate was 4·54 in 1911 and 5·91 in 1928, while the lowest and the highest rates in the intervening period was 3·71 and 5·59 respectively. The average rate was 4·71. In Queensland the rate in 1911 was 5·77 and in 1928 it was 6·37. It diminished to 4·16 in 1913 and rose to 6·36 in 1923. The average rate was 5·36. In South Australia the rate in 1911 was 5·79 and in 1928 4·73. The lowest figure in the intervening period was 3·97 in 1912 and the highest 6·88 in 1925. The average rate was 4·95. In Western Australia the rate in 1911 was 3·71 and in 1928 it was 6·66. The rate sank to 2·82 in 1921 and rose to 6·47 in 1925. The average rate is 4·86. In Tasmania the rate in 1911 was 4·6 and in 1928 it was 6·82. In 1919 it was as low as 2·64 and in 1925 it rose to 5·75. The average for the period is 4·66. For the Commonwealth of Australia the rate in 1911 was 5·03 and in 1928 it was 5·98. The lowest rate in the table is 4·27 for 1915 and the highest after the figure for 1928 was 5·92 for 1927. The average works out at 5·05. The rate in England and Wales in 1911 was 3·87 and in 1928 4·42. The lowest rate in the intervening period was 3·79 in 1917 and the highest was 4·37 in 1919. The average works out at 3·82.

Dame Janet Campbell admits that the mortality rate in England and Wales is considerably higher than it should be. It is still higher in every State of Australia. It will be noted that with the exception of South Australia the mortality rate in 1928 is higher than that of any previous year in the eighteen year period. This obtains as well for England and Wales. In other words the position has become worse instead of better. The rise in the rate is to some extent accounted for by an increasing accuracy in recording the cause of death and to a higher proportion of first births associated with a declining birth rate. But any inaccuracies traceable to these causes cannot account for the unsatisfactory state of affairs and the authoress admits that a stationary or even increasing death rate has to be faced.

In the next place she deals with the causes of the death of six mothers per thousand live births. In the ten years from 1919 to 1928 7,012 mothers died from causes associated with pregnancy and labour in Australia. Of this number 2,335 deaths were due to puerperal sepsis. This represents nearly one-third of the total number. The proportion is slightly higher in England and Wales. Accidents of pregnancy, including abortion, accounted for 1,003 deaths or approximately one-seventh of the total in Australia. The same causes were responsible for about one-tenth of the total number of deaths in England and Wales. Dame Janet Campbell calls attention to the high incidence of deaths from abortion and suggests that in spite of the many difficulties, it would be worth while to ascertain how many of the deaths were due to criminal interference. In regard to puerperal sepsis she refers to Professor Marshall Allan's report from which she gathers that, as is the case in other countries, this cause is more frequent in the large cities of Australia than in the small towns and country districts. She maintains that many of these deaths should be preventible. Puerperal convulsions, nephritis, uræmia and similar causes accounted for 1,450 of the deaths in Australia and 5,663 of the deaths in England and Wales. The proportion of the total in both countries is about one-fifth. Hæmorrhage and accidents of childbirth are the two next causes in the list. They accounted for one-eighth and one-tenth of the total number respectively. While hæmorrhage may be difficult to control as a cause of death, the group of eclamptic conditions and other accidents of parturition should be more susceptible to prophylactic measures.

The authoress further calls attention to the much larger amount of morbidity among women in pregnancy and childbirth in addition to the actual deaths. The illness and suffering resulting from the causes referred to above cannot be measured, although the records of the gynæcological wards of hospitals give some indication of this morbidity. In addition, there is the loss of infant life. These statements are platitudes, but Dame Jane Campbell finds it necessary to repeat the known in order to establish her case.

Infant Mortality.

Tabular statements of the number of deaths of infants in the first year of life in each State and in Australia as a whole are reproduced covering a period of twenty-eight years. It appears that for the Commonwealth the mortality has diminished from 96·91 per thousand live births in the quinquennial period from 1901 to 1905 to 52·96 per thousand live births in 1928. She holds that this is an achievement to be proud of. The death rates in Queensland of 45·54, in South Australia of 47·51 and in Western Australia of 48·14 are very encouraging. She finds that the conditions in Tasmania are less satisfactory. The death rate of 63·95 appears to her as unnecessarily high in a country where conditions appear to be favourable. While the death rate in the first year of life has diminished satisfactorily, the mortality among infants under one week has tended to rise. In 1907 the rate in Australia was 21·32 and in 1928 it was 22·19. The highest figure reached in the intervening years was 24·95 and the lowest 20·38. The highest rates are in Tasmania and New South Wales, while the lowest are in South Australia and Western Australia. The deaths in the first week are caused by obstetrical and developmental factors, while those in the later months are caused by pædiatric factors. She gives three principal causes of death as developmental causes, including prematurity, injury during birth, debility, congenital malformation, convulsions; respiratory diseases; gastro-enteritis. While debility, prematurity and convulsions are included in the first group, we wish to point out that these words convey little exact information. Debility may mean anything; prematurity connotes either a pathological process in the mother resulting in the early expulsion of the fœtus or a traumatic interruption of the pregnancy; convulsions are symptoms and not a disease. Until these terms are eliminated among causes of the death of infants, accurate information concerning the actual cause of death cannot be obtained. In ten years from 1919 to 1928 prematurity, injury at birth, congenital debility, congenital malformations and convulsions caused 38,764 deaths or 49·19% of all deaths under one year; gastro-enteritis caused 15,436 deaths or 19·59% and respiratory diseases caused 8,449 deaths or 10·72%. The deaths in the first group occur almost entirely during the first few weeks of life. Dame Jane Campbell states that some of the children are born without capacity for survival. She recognizes, however, that many of the deaths might be prevented by better midwifery, by supervision of the mother during pregnancy and by more skilful care of weakly infants after birth. She expresses the opinion that some of the deaths caused by respiratory diseases could be avoided if greater care were exercised to prevent infection from adults. She finds that gastro-enteritis is probably due to infection, though great heat renders the child more susceptible and less able to resist attack. Its prevention is a question of suitable and careful feeding. If weaning has to be carried out during hot weather, no trouble should be too great to obtain satisfactory milk and to preserve it from contamination. In the absence of reliable fresh milk, she recommends "a good brand of

dried milk." She warns against direct and indirect infection through flies.

She has been unable to obtain official information concerning the frequency of still-births, but accepts the figures given by Professor Marshall Allan of about thirty for every thousand live births. She points out that still-births represent an important loss of potentially viable children. In an English investigation of 1,083 deaths, 700 were deaths of infants and 383 were still-births. Of the 700 391 were deaths of infants during the first month of life, so that the still-births and the deaths of infants under one month represent three-quarters of the total wastage of infant life.

Infant and Child Welfare.

Before attacking the problem of examining the existing facilities for the care of infants and children in Australia, Dame Janet Campbell remarks on the similarity of the questions involved in maternal and child welfare in England and in Australia. She notes the advantages in Australia of a wholesome open-air life, of abundant sunlight, absence of much cold and damp weather, of good housing, absence of slums, of abject poverty and of overcrowding. The disadvantages are the great sparsely inhabited districts where the standard of comfort is low, where the water supply may be severely restricted, the dietary ill-balanced, the daily toil arduous and the medical, nursing and domestic help difficult to obtain. She speaks enthusiastically of the amazing capacity of the Australian woman to shoulder her burden.

Since instinctive knowledge of good mothercraft does not exist, infant welfare centres and trained visitors are needed. In New South Wales there are eighty centres under the immediate control of the Director of Maternal and Baby Welfare. There are 210 nurses appointed by the Department of Public Health. In Victoria there are two voluntary associations responsible for many infant welfare centres in addition to some independent centres. Support is given by the municipalities to the centres and the Government makes a substantial contribution toward the cost. The various activities are coordinated by the Director of Infant Welfare. In Queensland the Director of Infant Welfare is a part-time officer responsible to the Assistant Under-Secretary in the Home Secretary's Department. There are fifteen centres. The nurses are appointed by the State. In South Australia the centres are controlled by a voluntary body. They number forty-five. The work is subsidized by the State and municipalities. In Western Australia there are fourteen centres controlled by voluntary organizations. They are subsidized by the Government. In Tasmania the centres are in the control of voluntary bodies. The Government pays a subsidy and also appoints the nurses. The centres in all parts of the Commonwealth are good. The nurses are fully trained hospital nurses. Many are certificated midwives and some possess the certificate of one of the mothercraft training schools. The centres are visited by mothers of all social classes, unlike those in England. There is no "close medical supervision" which is regarded as essential in England. Dame Janet Campbell suggests that the reasons are probably that the mothers usually have their own family medical attendant and that there is difficulty in securing the services of a medical practitioner specially trained in infant welfare, who is not in private practice in the district. In addition to the baby centres, there are the baby hospitals and special wards in children's hospitals.

Attention is called to the gap in the provision for the child after it ceases to attend the centres and before it attends school.

Maternal Welfare.

The maternity hospitals in Australia as elsewhere have excellent prenatal clinics. Information is given concerning the department at the Queen Victoria Memorial Hospital in Melbourne. Prenatal work has scarcely been developed in connexion with the infant welfare centres. A few attempts have been made to see if prenatal clinics would be popular with the women, but "speaking generally there is no attempt at the systematic supervision of pregnancy by this means." Dame Janet Campbell continues:

> As regard private practice there is evidently a growing appreciation of the value of prenatal care and many doctors endeavour to persuade their patients to consult them as early as possible in pregnancy and to remain under regular observation, but much advance will have to be made before all doctors are willing and competent to undertake this duty and all patients prepared to cooperate with their doctors in the endeavour to prevent obstetric disaster.

Reference is made to the increasing accommodation for women during the period of parturition at maternity hospitals. Private maternity hospitals of all grades of efficiency exist in the majority of the towns. Dame Janet Campbell concludes with the remark that it seems likely that the supply of easily accessible maternity beds will keep pace with the growing demand for in-patient accommodation.

In regard to convalescent homes and after care of mothers, mention is made of the urgent necessity of making provision of this kind. The District Nursing Association in Melbourne instituted in 1926 an after-care home for patients who need convalescent treatment after discharge from hospital. Lying-in women are eligible for admission to the home. Many mothers derive benefit from the treatment. The Northcote Home near Adelaide has been established for the care of working mothers and their babies when rest and treatment are needed.

In isolated districts special arrangements are being made to provide medical, nursing and domestic assistance for the women. Improved transport service, including ambulance, has been instituted. The main arrangements have been carried out by the Bush Nursing Association and the Country Women's Association. The former exists in all States except South Australia. The primary object is to establish nurses in isolated country districts where medical aid is absent or difficult to secure rapidly. The nurse is prepared to undertake maternity work and to nurse patients in their own homes. Bush nurses are fully trained and possess midwifery certificates. Dame Janet Campbell gives a short description of the bush nurse's life and difficulties. Bush nurses are encouraged to exercise prenatal supervision of their patients and if any abnormal condition is suspected, the mother is assisted to enter the nearest maternity institution for her confinement. A short description is also appended of the Australian Inland Mission and the organized medical and nursing work conducted in Queensland under its auspices.

The Country Women's Association is now taking an active interest in matters connected with maternal and infant welfare, especially in New South Wales and Queensland.

Further Action.

Dame Janet Campbell states that in reviewing the maternal and child welfare work in Australia she has gained the impression that the time has arrived when effective development depends upon the securing of a policy in order that the work as a whole may be rendered efficient, more scientific and not unduly dependent on the good will, enthusiasm and generosity of individuals. It should take its proper place in organic relationship with all other branches of public health work. She therefore recommends improved professional education in midwifery and infant hygiene, improved legislative and administrative machinery, effective supervision of maternity and the development of the infant and child welfare services. She summarizes the courses of instruction in obstetrics at the University of Sydney. The Professor of Obstetrics at the University of Melbourne had not taken up his duties at the time of writing the report. She recognizes that the provision of adequate facilities for clinical study is often a real difficulty. She holds that the personal observation and delivery of twenty parturient patients recommended by the General Medical Council is the minimum consistent with effective teaching. If there is not sufficient opportunity at any training institution for the proper teaching of both medical students and midwives, the students should have the preference. She recommends that adequate training in the care and hygiene of the new born infant should be given to the medical student,

for the medical practitioner will ultimately assume the responsibility for the safety of the mother and the infant. She also advocates the provision of post-graduate facilities for practical work in obstetrics.

In regard to the training of the midwife, she finds that there is evidence of laxity of administration in some of the training schools, although the requirements in the several institutions throughout the Commonwealth are similar on paper. In the larger maternity hospitals the conditions are excellent, but in many of the smaller institutions recognized as midwifery training schools it must be impossible to carry out the requirements fully. The recognition of private maternity hospitals as training schools has obvious objections. After discussing the conditions she refers to the report on the training of midwives recently issued in England in which it was recommended that every pupil-midwife should undergo a course of three months' post-examination obstetric practice under supervision, during which time she should be made responsible for the conduct of not less than five labours before she is given a full licence for independent practice.

She deals in some detail with the requirements for the training of the midwife in mothercraft. Various schools have been established in the several States. She holds that it is eminently desirable that the training in mothercraft should receive full official recognition and support. As long as difference of opinion exists in regard to details of infant feeding and other matters connected with mothercraft, the instruction should be planned to allow of individual teaching of details.

Legislative Measures.

Dame Janet Campbell pleads for more uniform legislation governing the control of maternity problems throughout Australia. She finds that there is urgent need of accurate and comparable death returns. Greater precision is required in regard to the deaths attributed to puerperal fever. Common practice should be introduced in all countries. In regard to the registration of still-births, she states that since correct information is essential for any scheme for the reduction of neonatal deaths, this must not be overlooked. She wishes to adopt the definition of the League of Nations of the term still-birth. It is—

> the birth of a fœtus after twenty-eight weeks' pregnancy in which pulmonary respiration does not occur; such a fœtus may die either before, during or after birth, but before it has breathed.

She advocates the early notification of births, but recognizes the difficulties that exist in some of the thinly populated districts of Australia. It seems that there is an inclination to attribute the symptoms of puerperal fever to some other cause as long as possible and for this reason the notification of this infection as carried out in Australia at times causes confusion. In England the Ministry of Health decided in 1925 to make all puerperal pyrexia notifiable, so that doctors might be relieved of the responsibility of diagnosing puerperal fever. The definition of puerperal pyrexia is any febrile condition other than that notifiable as puerperal fever, occurring in a woman within twenty-one days of a birth or miscarriage, in which a temperature of 38° C. or higher is maintained for a period of twenty-four hours. In England the notifying practitioner is invited to state whether he wishes the assistance of the staff of the public health department, either in the form of bacteriological examination, of the service of a consulting obstetrician or of a trained nurse or of hospital accommodation. Even if the application of such a scheme in Australia might be difficult, its adoption in whole or in modified form is recommended. Dame Janet Campbell holds that it would be worth while to investigate the occurrence and effects of septic abortion.

Supervision.

The term maternity service connotes arrangements by which a woman may obtain the necessary facilities for the satisfactory conduct of her confinement. It involves not only attendance at the labour and ten days after, but also supervision throughout pregnancy and subsequent care of the mother and infant. In regard to antenatal supervision the medical examination should be made early in pregnancy and should include a full physical examination with a view to the detection of any general ill health or potential cause of sepsis. The urine should be tested, the blood pressure measured and the serum examined for signs of syphilis. A further examination should be made between the thirty-second and the thirty-sixth weeks of pregnancy to ascertain the presentation of the fœtus and the relation of the head to the pelvis. The urine should be tested at intervals throughout the period. The patient should be instructed in the care of her own health and in the preparation for the advent of her infant. The supervision may be carried out in the practitioner's consulting room or at a maternity hospital or at a clinic in connexion with an infant welfare centre. Dame Janet Campbell is of opinion that many practitioners are not fully convinced of the value of such systematic supervision, while many patients cannot afford private attendance of this kind. She points out that the antenatal clinic is the solution of this problem.

In dealing with the attendance at the confinement she points out that in Australia the great majority of women are attended by medical practitioners and comparatively few by midwifery nurses only. This is not the case in England. The relatively high maternal death rate in Australia and the excellent results obtained by competent midwives where they are largely employed, have led to the proposal that medical practitioners should leave normal midwifery to midwives and should be prepared to give antenatal advice and to render aid when difficulties arise. The advantages of the proposal are that the midwife gains great skill and experience, that she can keep free from infections, that she can bestow more time on her patient than can a medical practitioner, that she can give comfort and encouragement and by wise management persuade the mother to await patiently a natural issue to her pains, as she is not capable of employing operative measures, that she is to a large measure free from the importunities of the patient's friends. The one disadvantage of the proposal is that no one can anticipate when the labour will become complicated. Since Australian mothers seem to prefer to have a medical practitioner to attend them, Dame Janet Campbell thinks that it would probably be wise to effect a compromise. The medical practitioner should accept the full responsibility for every pregnancy and parturition, but may leave the actual delivery to the trained midwife when the conditions are normal.

A few words are devoted to the necessity under existing conditions in Australia for some confinements to be conducted in the patient's own homes. Otherwise obstetrical work is carried out either in a large maternity hospital, in a special ward of a general hospital or in a private hospital. The obvious advantages of the large institutions are that the services of special departments are at all times available. The smaller public hospitals and the private institutions may require enlightened supervision and control which administrative medical officers trained in maternity work should be in a position to give. Experience of outbreaks of puerperal sepsis in small maternity hospitals in England has convinced her of the importance of maintaining a prescribed standard of equipment and staffing, of preventing habitual over-occupation and of devoting meticulous attention to every detail of obstetric technique. She would revise the conditions under which private hospitals may be registered. The whole control should be in the hands of a responsible authority. She recognizes the advantages of establishing intermediate wards in large public hospitals.

In regard to the after care, she recommends that all women should be examined by a medical practitioner six weeks or two months after the confinement in order to insure that the recovery is complete and that no unrepaired injury remains to cause physical disability.

Infant and Child Welfare Services.

The object of infant and child welfare services is to give the mother sound advice concerning the care of the normal baby and little child under school age, with a view

to the maintenance of health and the prevention of illness. Voluntary bodies have achieved a great deal in this direction. It has, however, to be recognized that voluntary bodies cannot plan and carry out a national service. The responsibility for organization and control must be in the hands of the local authorities. The latter are glad to utilize the services of the voluntary organizations. In Australia the voluntary societies must sooner or later come under the supervision and control of an official authority which will be responsible for the general policy and for the greater part of the cost of maintenance. She suggests that the voluntary bodies should have reasonable freedom to carry on and extend the work of the centres, but should be required to cooperate with each other more than they do. New centres will have to be established. The existing overlapping should be eliminated and more unity of effort achieved. The centres should be placed under medical supervision. This may not be immediately practicable, but it is essential. Medical control cannot become effective without the active support of the medical profession.

> Until practitioners are prepared to take the trouble, not only to understand the functions and aims of the welfare centre and to realize that it is designed to be a valuable auxiliary to general practice and not a competing agency, but to acquaint themselves personally with the methods of natural and artificial feeding in which the nurses are well trained, there can never be the freedom from suspicion and mistrust, much less the whole-hearted cooperation, which is so essential.

In regard to the preschool age, she finds that periodic supervision by a medical practitioner is essential. The nurse is not qualified to give this service. Systematic home visits by the nurse are valuable if supplementary to medical examination. She advocates the carrying out of treatment of defects discovered at the medical examination either by the private doctor, by the hospital medical officer or possibly by a medical officer in attendance at treatment clinics. Dental supervision should be commenced before the child goes to school. It is also necessary to remember that the teeth of the pregnant or nursing woman must receive attention.

One of the difficulties encountered in the planning of a general scheme of maternal and child welfare is the incomplete organization of the public health service and the differences in administration in the several States. In England the matter is relatively simple. One of the branches of the medical department of the Ministry of Health is concerned solely with maternal and infant welfare. The local authorities are responsible for the health work carried out in the area which they represent. She thinks that it would be advisable if a properly organized division of maternal, infant and child welfare were established in each State as a branch of the public health department in charge of a whole-time medical director responsible to the chief medical officer. The division should be made responsible for the control of all matters concerned with the medical and social welfare of expectant and nursing mothers and of children under school age. Gradually the allied organizations could be brought under the single control. The director should be possessed of capacity for administration, beside having high medical qualifications and he or she should have practical knowledge of the work already done. The director should be prepared to cooperate with the medical profession. An advisory council representative of the several interested bodies might also be set up. A suitable scheme ultimately to be adopted, should be devised when the maternal and child welfare division is in existence. Among the matters with which the division would concern itself, are the conditions of appointment of the staff engaged in infant and child welfare work, the qualifications, salaries and conditions of service of each officer, whether further training is necessary in mothercraft and public health nursing and the like. She holds that the division should aim at uniformity of method. The division should be in close contact with all other departments and organizations concerned with work allied to maternal and child welfare, such as hospitals, the State children's boards, the school medical service and so forth. She states that the principle that the acceptance of a subsidy from the State for the support of any particular branch of work should carry with it certain obligations in regard to the expenditure of money granted, might receive full recognition. The contributions from the State Governments will have to be considerable, but support will readily be given to a scheme designed to save women from risk to life and health during child bearing.

She devotes some space to the question of publicity and propaganda, to the teaching of mothercraft in schools and to the influence of women's organizations on the work. In regard to research she advocates investigations into the causes of maternal death, into the physiology of pregnancy and into many other problems connected with her subject.

In summarizing her views on the administrative matters, she expresses the opinion that the Commonwealth Department of Health should establish a division of maternal and child welfare for the collection of information and for the extension of assistance. It is her considered opinion that a medical woman should be appointed director of the division. Subsidies should be granted by the Commonwealth Government to the States on defined conditions and grants should be given for research. The Federal authority should convene annual conferences. It should be responsible for the framing of the syllabus for nurse trainees and for the fixing of the standard of examination entitling to certification. The arrangements for continued experience after certification (Dame Janet Campbell uses the term post-graduate; there are no degrees granted to nurses and in consequence the term is not admissible) should also be a function of the Commonwealth division.

Conclusions.

Dame Janet Campbell's main conclusions are that the Governments of Australia must be prepared to expend more money than they are doing on maternal and child welfare, that the money provided should be economically administered, that special departments should be set up and should assume proper responsibility for this important branch of public health administration and that voluntary agencies should prepare themselves to surrender some portion of their independence.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION IN CONJUNCTION WITH THE CLINICAL SOCIETY OF THE MATER MISERICORDIÆ HOSPITAL was held at the Mater Misericordiæ Hospital on October 15, 1929, DR. E. D. AHERN, the President of the Clinical Society, in the chair.

Injury to the Scrotum.

DR. A. S. ROE presented a young man who had been injured while at work. His scrotum had been caught in some machinery. Half of the scrotum had been torn off and the remainder had been left hanging by a tongue of skin a few centimetres in width. Three-quarters of the skin of the penis had also been torn. There had been considerable shock.

Mercurochrome in 1% dilution had been applied and the patient had been anæsthetized a little later when an attempt had been made to cover the denuded parts. A strip of skin had been taken from the top of the remaining piece of scrotum and had been wound around the penis. This strip had been sewn to the remaining skin of the penis. The remainder of the detached scrotum had been pulled across to cover the two testicles which were hanging free by their cords. Two small drainage tubes had been left under the flaps. The parts had been dressed regularly with mercurochrome. The patient had made an uninterrupted recovery. Very little suppuration had occurred.

Rupture of the Bladder.

Dr. Roe also showed a young man who had been admitted to hospital for a very severe injury caused by a fall from a horse while he had been under the influence of alcohol. No reliable history could be obtained, but the patient assured Dr. Roe that he had emptied his bladder shortly before the accident. He had been suffering from shock. His abdomen moved, but had been held on guard. There had been tenderness over the region of the right kidney. A well defined fluid swelling, not coloured, had been noted over the pubes. Movement of the left leg caused pain and signs of shock; the patient appeared to be unable to define the nature or situation of the pain. A soft catheter had been passed without difficulty and a small quantity of blood-stained urine had been withdrawn. The patient had been admitted at night time; in the morning his condition was worse. X ray examination revealed a fracture of the pelvis; the tip of the catheter was seen to be lying in the abdominal cavity.

An operation had been performed. A large tear had been found in the posterior wall of the bladder above the level of the ureters. This was sewn up with interrupted catgut sutures and the abdomen had been swabbed. The bladder had then been opened and the tear had been sewn from the inner side. A self-retaining catheter had been secured in the bladder and a drainage tube left in the abdomen. Dr. Roe stated that in similar conditions he would sew up the abdomen without drainage. The patient had made a good recovery.

Abscess of the Lung.

DR. ELLIS MURPHY showed a married woman, aged forty-one years, who had had several teeth extracted under a general anæsthetic eight weeks before admission to hospital. She had stated that a considerable time had elapsed before she had regained consciousness after the operation and that she had vomited several times. Two days later she had felt ill. There had been a sharp pain in the right axillary region and a little fever. Several days later she had coughed up some foul smelling pus. The cough had persisted and there had been a moderate amount of offensive sputum, more abundant in the mornings. She had had several rigors and a few attacks of slight hæmoptysis.

On admission the patient had appeared to be bright. She had been very thin and had an irritable cough with fetid sputum. The trachea had been felt a little to the right of the middle lone. The apex beat had been in the fifth space eleven millimetres inside the nipple line. The heart sounds had been clear. The movements of the right side of the chest had been restricted. There had been diminished vocal fremitus and decreased resonance of the percussion note over the lower half of the right lung and diminished air entry in the same area. The sputum had not contained any tubercle bacilli. X ray examination had disclosed some fibrosis in the region of the right middle lobe with a rounded shadow suggestive of a pulmonary abscess and dislocation of the mediastinum to the right. No foreign body had been detected.

Several days later fifteen cubic centimetres of "Lipiodol" had been injected into the bronchial tree on the right side by the tracheal method. A skiagram had revealed a wedge-shaped area corresponding to the middle lobe on the right side into which no "Lipiodol" had entered. The patient's general condition had become excellent and her temperature had been practically normal. No operative treatment had been considered necessary. Rest in bed and a mixture of creosote and potassium iodide had been prescribed. The patient had improved and at the time of the meeting there was no sputum, the temperature was normal, she had a good appetite and slept well, but from time to time complained of a little pain in the right axilla.

Empyema.

Dr. Murphy also presented a male patient, aged thirty-nine years, who had been admitted to hospital with acute lobar pneumonia of the lower lobe of the right lung. The temperature had fallen by lysis and had then become irregular. Signs of fluid in the pleural cavity had appeared. An operation had been performed and some pus had been removed. The patient's condition had improved for several days and had then become worse and evidence of septic absorption had become apparent. The pulse rate had varied between 115 and 128 in the minute; there had been intermittent fever reaching 39° C. (102·2° F.). Sweating had also occurred. The patient had been losing weight. X ray examination had revealed shadows at the base of the right lung, obliterating the costo-phrenic angle, and also a shadow in the region of the interlobar septum between the lower and middle lobes on the right side. Attempts had been made to explore the region with an exploring syringe, but no pus had been obtained. A further attempt had been made higher up some days later, but still without success. The patient's condition had become steadily worse. It had therefore been decided to explore the pleural cavity again. Dr. Hemsley had carried this out while the patient was under chloroform anæsthesia. The whole cavity had been explored, but no definite pocket of pus had been discovered. Tubes had been left in the opening. Several days later there had been a flow of pus from the wound. The patient had gradually improved and had been able to leave the hospital on the day of the meeting. Dr. Murphy stated that the history of this patient's illness illustrated the very great difficulty that sometimes occurred in the localization of collections of pus in the thoracic cavity and the necessity for continuous and thorough exploration.

Extra-Pyramidal Rigidity.

DR. JOHN BOSTOCK introduced a patient, a male, aged twenty-two years, who had complained of "shaking all over" for a period of nine months. Locomotion had become difficult and he had been incapable of working. The diagnosis of extra-pyramidal rigidity had been based primarily on the cog-wheel phenomenon of the wrists, the obvious spasticity of the muscles, with comparative absence of signs pointing to pyramidal involvement. The condition was due to disseminated sclerosis. The blood serum and the cerebro-spinal fluid had been examined, but no Wassermann reaction had been obtained. Examination of the blood had not revealed any abnormality. The liver function tests had yielded normal responses, indicating that the hepatic efficiency was unimpaired. Dr. Bostock said that there was a possibility that this condition was part of a progressive lenticular degeneration and that it was associated with changes in the liver.

Post-Traumatic Hysteria.

DR. K. C. ROSS gave a demonstration on the condition of a male patient, aged twenty-three years, who had been struck on October 3, 1929, by a falling packing case on the right clavicle. This had been followed at once by complete loss of movement of the right arm. When the patient had been seen on October 5 he had complete loss of movement of all the muscles of the right upper limb and its girdle, but the unsupported forearm had remained at 90° flexion, in the position of an arm in a sling; passive movements had been strongly resisted. Sensation had been completely lost below a line encircling the limb at the outer border of the acromial process. The hand had been bluish and considerably colder than its fellow. He had complained of tenderness immediately above the middle of the clavicle, but there had been no evidence of trauma. Three days later he had been able to flex and extend the distal two joints of the thumb and had complete return of sensation below the intercarpo-phalangeal joint of that digit. Otherwise the condition had been unaltered. He had been seen again on October 12, when he had regained sensation in the whole limb and could move all his fingers to a slight degree. He still maintained that he was quite unable to move any other portion of the limb either passively or actively, but while he was pulling off his singlet and putting it on again he moved both the arm and the forearm to a slight extent. At the time of the meeting there was a moderate degree of movement at the wrist and very slight pronation and supination. He still resisted passive movement and all attempts to move the forearm, even with the elbow supported, caused him to complain of pain at the site of the injury. Adjustment of his clothing was accompanied by still more movement at the elbow and shoulder. There seemed to be little doubt that the lesion was entirely an hysterical one and that recovery would be complete and rapid.

Ciliary Staphyloma.

DR. ALEX MACDONALD presented a man, aged sixty-eight years. He had been seen for the first time in December, 1925, when he had stated that he was a drover and had suffered from pain and swelling in the right eye and loss of vision on the right side. The symptoms had been present for three months. He had attended at the Brisbane Hospital for three weeks in November, 1925, and had been told that he had a tumour of his eye and that the eye would have to be excised. Examination had revealed that the tension of the eye was normal, that the pupil was oval and drawn downwards and outwards, that there was ectasia of the sclera on the outer side in the ciliary area, that the vision of the right eye was $^{6}/_{60}$ and of the left eye $^{6}/_{6}$, that on transillumination the ectatic area seemed to be clear and that the discs were normal. The patient stated at a later date that the condition had remained unchanged. After five weeks' treatment the staphyloma had receded, the pupil had become round and had assumed the normal position and the tension had remained normal. The patient had been brought to a meeting of the Eye, Ear, Nose and Throat Section of the Queensland Branch in February, 1926; the consensus of opinion had been that the condition was due to a melanotic sarcoma of the ciliary body or chorioid and that the eye should be excised. Dr. MacDonald had decided to keep the patient under observation. The condition of the eye had improved and the vision in 1928 had been $^{6}/_{36}$ without the aid of lenses. By 1929 the ectatic condition had become less, all pain and irritation had disappeared and the vision had improved to $^{6}/_{24}$ unaided. The conclusion he had formed was that the condition had probably been caused by a thinning of the sclera with consequent showing through of the underlying pigment.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Salisbury, Charles Victor, L.R.C.P. (Edinburgh), 1928, L.R.C.S. (Edinburgh), 1928, L.R.F.P.S. (Glasgow), 1928, Glenhope, Pennant Hills.

LISTS OF MEMBERS.

THE lists of members of the several Branches of the British Medical Association in Australia are now in the press and will be available in a short time. Copies can be purchased from the office of THE MEDICAL JOURNAL OF AUSTRALIA at one shilling each.

Medical Societies.

THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN was held at the Hospital for Sick Children, Brisbane, on August 22, 1929, DR. D. GIFFORD CROLL in the chair. The meeting consisted of a series of clinical demonstrations.

Fragilitas Ossium.

DR. D. GIFFORD CROLL showed a girl, aged fifteen years, who was suffering from *fragilitas ossium*. She had been shown at previous clinical meetings over a number of years. The child's father said that at birth she had had a shiny lump on the left thigh. This had been examined by X rays at the Royal Prince Alfred Hospital, Sydney, when the child was fourteen days old and the condition had been regarded as a probable fracture. The father stated that every time new teeth appeared the lump had got larger and shiny and the child had complained of pain. In the intervals of time between the eruption of teeth the shininess had disappeared and there had been no pain. When the child was one year and six months old the left femur had become fractured at that spot and since then the child had had twenty fractures, only two of these being in places other than the lower limbs. One fracture had occurred in the left arm and the other in the left cavity. The last fracture had occurred two and a half years previously and had been in the left thigh. According to the family history the mother had had five children with one miscarriage between the second and third children, two years after the birth of the second child. The patient shown was the second child. The eldest child, a girl, aged sixteen years, had had seven fractures, all in the left thigh except the last but one which had been in the left tibia. The eldest child had had no fracture since she was seven years old and the only sign of fracture was a small amount of callus on the left shin. The third child, a boy, had been born four years after the patient and was normal. A girl, born two years later and a boy born six years later were normal. There was no history of this disease in any other branch of the family. All the members of the family had been subjected to the Wassermann test and no reaction had been obtained.

It was pointed out that the patient stood in her boots with the left side of her pelvis tilted in an upward direction. Definite scoliosis was present and the father stated that the "right-sided bump" had developed since the occurrence of the last fracture three years previously. The space between the right iliac crest and the lower border of the ribs was broader than that on the left side. The right gluteal muscles were more developed than the left and the right shoulder was held higher than the left. The right arm was slightly longer than the left. The right leg was 2·5 centimetres (one inch) longer than the left. The patient was wearing on the left foot a boot with a cork inlay five centimetres (two inches) high; this, according to the father, had been ordered three years previously at the Hospital for Sick Children. *Genu valgum* was pronounced in both lower limbs, but was worse in the right and this had appeared quite recently, making the present difference in the length of the limb only 2·5 centimetres. The power in both quadriceps muscles was good. The right tibia was considerably deformed and rotated in an inward direction and as a result of the *genu valgum* the right foot was thrown outwards. In the left tibia which had not been fractured, considerable anterior bowing was present. The child's skull had the peculiar shape associated with the disease; it was broad and large and had an angular projection running round the posterior and lateral aspects just above the level of the auricle. There was a small upper and a relatively long lower jaw and the nose and chin were pointed. Intellectual development was normal.

Dr. Croll said that Dr. McDowall had made an X ray examination of the bones and thought that they were undergoing a softening process which accounted for the twisting and deformity which were increasing.

Diabetes Mellitus.

DR. H. MATHEWSON showed a child who was suffering from *diabetes mellitus* and who had been shown at previous meetings. An X ray examination of the skull had disclosed no abnormality. The carbon dioxide combining power was found to be 38. Since the last time that the patient was shown the amount of "Insulin" had been increased by one unit a day to twelve units twice a day. The figures obtained on blood sugar estimation were 0·72 and 0·85 half an hour after a meal containing 17·5 grammes of carbohydrate. Nutrition was good and the child was bright. Dr. Mathewson said that he intended, if necessary, to increase the "Insulin" still more.

Erythrœdema.

Dr. Mathewson also showed a child, aged ten months, who was suffering from erythrœdema. The rash on the hands and feet was pronounced, but the body was free from eruption and the child did not scratch itself. Photophobia was not pronounced. The child was sleeping badly

and was not taking its food well. During the last week it had lost 0·45 kilogram (one pound) in weight. The condition had gone on for three months. The only drug given had been large doses of recently prepared calcium and the ultimate outlook was good.

Elephantiasis.

DR. F. W. LUKIN showed a patient, aged ten years, who was suffering from swelling of the left leg of twelve months' duration. Three years previously she had fractured her left femur and it had been refractured on being taken out of an extension apparatus six weeks later; good union had resulted after three months. Eighteen months previously three distinctly red raised patches had appeared on the left leg, two below and one above the knee joint. The condition had been diagnosed as erysipelas and had disappeared within five days of the application of ichthyol and glycerin. The leg had been swollen ever since and apparently had become more swollen than usual in the preceding five weeks. A test for filaria had been carried out on August 21, 1929, but no sign of filaria had been discovered. Dr. Lukin thought that the condition was early elephantiasis. The child had lived in Papua and was alleged to have suffered from malaria and filaria while it was there.

Cyst of the Leg.

DR. S. F. MCDONALD, on behalf of Dr. G. P. Dixon, showed a child, aged seven years, who complained of a swelling behind the left knee. The swelling had been present for two weeks and no response had followed the application of a test for filaria. There was neither pain nor discomfort. Examination revealed a painless cystic swelling in the left popliteal space and more to the left side. The swelling was not attached to the skin, there was no pulsation and the swelling appeared to become smaller on flexion of the knee joint.

Œdema of the Glottis.

DR. C. WASSELL showed a child, aged one year and three months, who had been admitted to hospital on August 2, 1929, with the history that the evening before he had become cross and irritable and the voice had changed. The child had been seen in the out-patient department and a serious view had been taken of the condition. Hoarseness and dyspnœa had been present and a diagnosis of œdema of the glottis had been made. Treatment had rested between intubation and tracheotomy and the latter had been performed, for it was thought that with the former redundant mucous membrane might project over the tube. The child had been put into a steam tent and general treatment had been instituted. One week later some stertor had appeared; this had lasted for a day or two and had disappeared. The diagnosis as to the cause had been difficult. The œdema had been definitely inflammatory and probably either influenzal or diphtheritic Three swabbings had been taken, but no causative organism had been discovered. The appearance of the epiglottis was that of a large cherry projecting upwards on the back of the tongue. The prognosis was grave.

Papilloma of the Larynx.

Dr. Wassell also showed a child, aged five years, who had been admitted to hospital on February 13, 1928. Tracheotomy had been performed immediately and the tube worn for thirteen months. A laryngoscopic examination had revealed the presence of papillomata of the larynx. The child had been kept a considerable time waiting for the radium clinic. On March 9, 1929, two radium needles had been inserted down the trachea between the vocal cords. They had been allowed to remain in this position for two to three hours. Two needles had then been suspended between the cord for twenty-four hours. The tracheotomy tube had been left in position for a further period of six weeks and had then been removed. There had been no obvious benefit from the radium. The tracheotomy wound had been allowed to close up, but three weeks perviously the child had become so distressed that the tube had been hurriedly reinserted. Dr. Wassell laid stress on the length and tediousness of the treatment.

Correspondence.

SUPERFŒTATION.

SIR: A report from Dr. Willis in the journal last April makes the following incident worth recording.

This morning at three o'clock I was called to see a young married woman, the mother of three children, who her husband said was "having a miscarriage." I found her sitting on a chamber in the kitchen, pale from loss of blood. She "had had pains all night and had just passed something." I sent her to bed and meanwhile with the poker explored the contents of the chamber I fished out a fœtus in its sac complete. The sac would be of about one ounce in content and had the usual appearance of an early complete abortion. Congratulating myself on the prospect of an immediate return to my couch, I approached the patient with confidence, but to my surprise found suspended from the vagina a well developed fœtus about four inches in length. Attempts to express the secundines were not successful, so I broke off the cord and some twelve hours later a well formed placenta was removed under general anæsthesia.

Yours, etc.,
ARTHUR E. PANTING.

Launceston, Tasmania.
February 5, 1930.

DECENTRALIZATION OF MATERNITY HOSPITALS.

SIR: Despite much effort in the form of investigations into the high rates of maternal mortality and morbidity there is little variation in their statistics.

Apart from stressing the need for antenatal supervision and more or less uncomplimentary references to half-trained medical practitioners, our leading obstetricians have put forward few practical suggestions. The problem is generally attacked with regard to the larger centralized or city hospitals. Let us for a moment consider the work done in the whole of the country districts of Australia, where many thousands of confinements take place annually. Here, I am sure, if statistics could be obtained, the percentages of maternal mortality and sepsis would be surprisingly low. It would therefore appear more rational to provide accommodation at certain accessible country-side towns and so discourage the flow from the country districts to the larger centralized city hospitals (as well as to the smaller private hospitals) where, despite a variable degree of care, there lurks the greatest cause of maternal mortality—sepsis. Rather would I suggest that city dwellers leave for the country.

Dr. Henry Jellett states that "80% to 90% of women deliver themselves." Surely we can decrease the appalling mortality by permitting them to deliver themselves in cleaner towns and purer air.

Yours, etc.,
A. A. HILL, M.B., B.S. (Melb.).

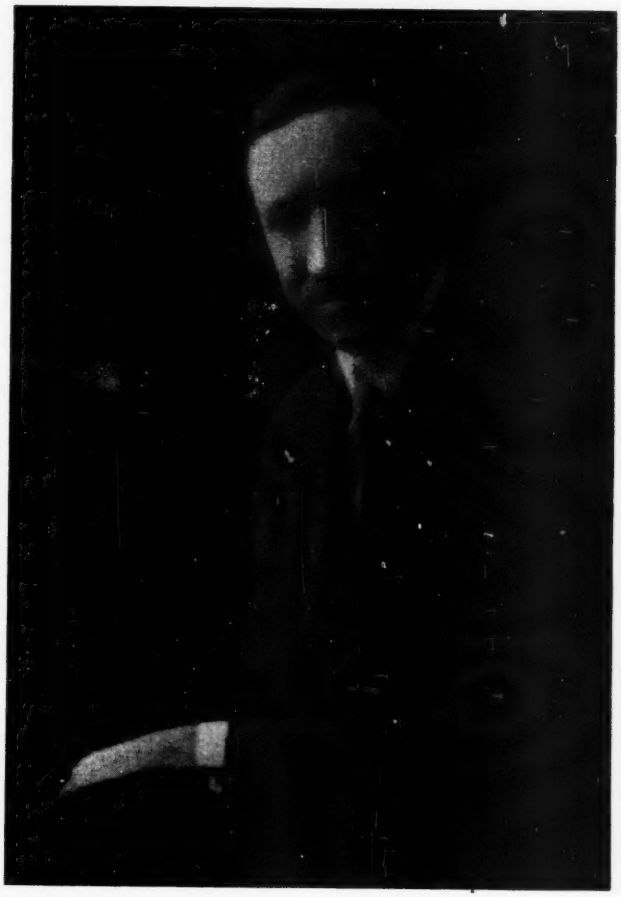
Kellerberrin,
Western Australia.
February 3, 1930.

Obituary.

THORBURN BRAILSFORD ROBERTSON.

ON January 18, 1930, Professor Thorburn Brailsford Robertson died at a private hospital in Glenelg, near Adelaide, after a short illness. He contracted influenza a short time before his death, but returned to his work before he had recovered from the infection. His lungs became affected and after a few days he died of post-influenzal pneumonia. His death is a very serious blow to the University of Adelaide and indeed to the world of physiology.

Thorburn Brailsford Robertson was the son of Thorburn Robertson, a mining engineer. He was born in Edinburgh in 1884. Ten years later his parents moved from Scotland to South Australia. The boy was sent to a private school at Glenelg. In due course he passed the matriculation examination and entered the University of Adelaide. He was then eighteen years of age. In working for his degree in science he displayed unusual ability and gave evidence of originality. At the age of twenty-one years he obtained the degree of bachelor of science with first class honours. In May, 1905, he was appointed assistant lecturer in physiology at the University of California under Professor Jacques Loeb at a time when the latter was conducting his tissue culture experiments and carrying out his extraordinary work on the fertilization of the ova of echinoderms, *Chætopterus*, marine molluscs and other forms of life. It will be remembered that Loeb started his work on artificial parthogenesis in 1891 or 1892 and that he published a collection of twenty-five papers in 1906, at the time when Thorburn Brailsford Robertson was working under his guidance. Robertson found his chief stimulating, helpful and encouraging and Loeb found his pupil keen, ingenious and painstaking. There existed in those days some bitter antagonism against Loeb, particularly on account of his attitude toward the work of Charles Darwin, but also because of a mistaken conception of Loeb's objective. Robertson adopted Loeb's outlook on the nature of cells and living matter. He thus strengthened his position in the department at the University of California. In 1907 he obtained the degree of Ph.D. and in the following year the University of Adelaide granted him the degree of doctor of science. In January, 1908, he was appointed Assistant Professor of Biochemistry and later in the same year Assistant Professor of Physiology. About this time he devised a special extract of the anterior lobe of the pituitary gland which he found exercised a definite influence on growth. The extract was named "Tethelin." This substance was patented. The work thus begun was continued for many years by him and has been fruitful in throwing light on the processes of growth and the healing of wounded tissue. Loeb accepted the position of member of the scientific staff of the Rockefeller Institute for Medical Research in charge of experimental biology in the year 1910. Brailsford Robertson was then promoted to the position of Associate Professor of Physiological Chemistry and Pharmacology. He remained in this position until 1918 when he was offered and accepted the position of Professor of Biochemistry in the University of Toronto. He remained in Toronto for only one year. The death of Edward Charles Stirling on March 20, 1919, rendered vacant the chair of physiology at the University of Adelaide. It was offered to Thorburn Brailsford Robertson and he entered upon his duties in the same year. From the first he set to work to remodel the department. Biological chemistry occupied his first attention. He found it advisable to recast the course in physiology and to apportion the teaching to various demonstrators. He continued to prosecute his researches on growth. Early in 1922 when Banting and Best announced that a substance known as "Insulin" had been isolated from the islands of Langerhans and that this substance was capable of controlling the sugar content of the blood in diabetes, an urgent demand arose in Australia for supplies. The Commonwealth Serum Laboratories were not able at first to meet this demand. Brailsford Robertson immediately stepped into the breach and offered to prepare "Insulin" under the conditions prescribed in the letters patent. Arrangements were concluded rapidly and the extract was carefully and well prepared under his control.



Thorburn Brailsford Robertson was a first class lecturer and an interesting teacher and in consequence soon became very popular with the students under his care. Although medical students are not usually interested in experimental work into the processes of growth and cannot be expected to find biochemistry a fascinating study until they learn to apply its doctrines to the diagnosis and treatment of disease, Robertson had no difficulty in surrounding himself with a small band of enthusiastic workers who had had their keenness aroused by their admirable teacher. The students realized from the first that their new professor was working to improve the department of physiology and the medical school of their university. It happened that shortly before he came to Adelaide the trustees of the late John Darling determined to erect a memorial in the shape of a building attached to the medical school. The building was started after Robertson had arrived. It was he who undertook the selection of the equipment and fittings. It was he who influenced the architects in their designs of the laboratories and store rooms. The Darling Building was opened on May 15, 1922, and from that time the medical school became transformed.

On April 16, 1920, a meeting of medical practitioners was held at the University of Adelaide for the purpose of inaugurating a society to be known as the Medical Sciences Club of South Australia. At that meeting Brailsford Robertson took a leading part. The meeting appointed Dr. H. Swift the chairman and Thorburn Brailsford Robertson the honorary secretary. Since that time frequent meetings have been held and many important communications have been read by its members. Robertson was a frequent and a very valuable contributor. Some three years later Miss A. F. Keith Sheridan having been attracted by the scientific researches that Robertson was

prosecuting at the University, made a substantial bequest to the University for the purpose of medical research. Out of this fund Brailsford Robertson and Professor J. Burton Cleland established a journal known as *The Australian Journal of Experimental Biology and Medical Science*. The first issue was published on March 15, 1924. It will be noted that the title of this journal has a similarity to that of the *Proceedings of the Society for Experimental Biology and Medicine*, a New York publication of which Robertson had a high opinion. He himself was a member of the New York Society. In the first issue of the new magazine Robertson published an article on the influence of hydrolysis upon the capacity of proteins to bind acids and bases. His publications in this and other journals have been many and have covered a large range of biochemical and biological subjects. In 1924 he worked on several problems connected with the culture of the infusoria. He then attacked the subjects of the function of the lipoid in mitochondria, the permeability of polarized membranes and the utilization of nucleic acids of animal and vegetable origin. It is not possible to refer in this place to all the matters studied by him. His main objective was to gain a fuller understanding of growth as a physical and chemical property. This work involved a very large series of breeding experiments and an endless endeavour to create modifications in cellular growth and development by chemical and biological means. In 1926 the journal underwent a material change, in that its endowment was altered. Sir Joseph Verco placed the sum of five thousand pounds in trust with the Council of the University of Adelaide and directed that the income was to be used to sustain the journal. The journal thus became the property of the University.

In the year 1927 Thorburn Brailsford Robertson was appointed a member of the Australian Council of Scientific and Industrial Research and was placed in charge of the animal nutrition laboratories that had been erected by the Council in Victoria Drive, Adelaide, for the purpose of applied research into the problems of growth and nutrition in animals. This appointment involved a considerable amount of planning and organization. Robertson was in his element and as usual achieved signal success in the founding of the laboratory in which he conducted his further investigations on growth. While he worked experimentally in his laboratory, others applied the results in field stations in different parts of the Commonwealth. It will thus be realized that the work begun under Loeb in the University of California was continued and extended until at the time of his death Thorburn Brailsford Robertson was Professor of Physiology at the University of Adelaide, teaching, lecturing and organizing the work of his department, editor of an important scientific journal, head of a semi-official laboratory at which experiments having a great economic significance were being carried out, and leader of an earnest band of scientists at the University engaged in biochemical research. He was a man who always found time for any important piece of work and astounding as it may seem, he also found time to indulge in extra tasks, such as writing text books, story books for children and the like.

His preeminent place in the world of science was recognized as it was bound to be. In 1926 he was elected a foreign member of *L'Accademia nazionale dei Lincei*, a distinction of moment. He was a member of many scientific societies in Australia, England and the United States of America.

Thorburn Brailsford Robertson married the third daughter of the late Edward Sirling in 1910. They had two sons and one daughter. In his home life he was truly delightful. A lover of children, he brought all his gifts and gentleness to colour the lives of his own offsprings. In private life he was courteous, charming and amusing. Although he was always enthusiastic about his work, he could divorce himself from his daily vocation and take pleasure in the small things of life. His philosophy was healthy and encouraging; he had a broad mind and was tolerant in his judgement of others. What he has started in Adelaide will not cease to be; his influence was so profound that those who came under the spell, will carry on his ideas and march steadily along the road he cleared with such erudite anticipation.

ARTHUR MACKENZIE WILKINSON.

IT is with regret that we have to record the death of Arthur Mackenzie Wilkinson which occurred at his home, Glenferrie Road, Hawthorn, Victoria, on November 7, 1929.

Arthur Mackenzie Wilkinson was the second son of the late H. J. Wilkinson, for many years incumbent of Saint George's Church of England, Queenscliff, Victoria. He was educated at Geelong College and entered the Melbourne University in 1884. After graduating M.B., Ch.B. he served for a term as resident medical officer at the Melbourne Hospital. He was closely associated in his professional work with Sir Henry Maudsley, Sir Richard Stawell, Dr. Julian Smith, Dr. S. V. Sewell and the late William Moore. Shortly after completing his post-graduate studies he commenced practice in Hawthorn where he remained in active practice until the time of his death forty-two years later.

Arthur Mackenzie Wilkinson's chief recreation was in his garden and he was a great lover of roses, but his real hobby was his work and he gave his whole life to the service of his patients, by whom he was loved and trusted.

His passing came as a great sorrow to many friends both among his colleagues and among those whom he had served so well. He leaves a widow and two married daughters to whom our deep sympathy is extended.

Dr. Julian Smith writes:

I knew him for many years. He began life as a doctor on the Mornington Peninsula; then he went abroad for study and on return settled where he died. From the beginning he set himself the highest ethical professional standard and refused all lodge work. He always recognized the great responsibility of a family physician and zealously kept himself informed of all advances in our craft. He was a mighty and omnivorous reader of all medical literature, usually into the early morning hours.

His profession was his hobby and his patients recognized this and remained loyal to him to the very end. His calm courage in the face of his approaching end and his steadfast adherence to duty in face of all protest till sheer exhaustion laid him low, were alike remarkable. Of no man can it be better said that "he went about doing good" and his passing is universally regretted.

University Intelligence.

UNIVERSITY OF SYDNEY.

In our issue last week we announced the appointment of Dr. Charles George Lambie, M.C., F.R.C.P. Edinburgh, F.R.S. Edinburgh, as Bosch Professor of Medicine, of Dr. Harold Robert Dew., F.R.C.S. England, as Bosch Professor of Surgery, and of Dr. Hedley Duncan Wright. D.Sc. (Edinburgh), as Bosch Professor of Bacteriology. We have received the following information from the Senate of the University.

Sixteen applications were received for the chair of surgery, seventeen for the chair of medicine and fourteen for the chair of bacteriology. The applications were first considered by committees of advice in London, consisting of experts in the several branches of medical science. The recommendations of the home committees were unanimously adopted by both the special committee in Sydney and by the Senate.

Professor C. G. Lambie graduated M.B., Ch.B. in Edinburgh in 1914 and proceeded to the degree of M.D. with first class honours and the gold medal in 1927. He is thirty-eight years of age; is a Fellow of the Royal College of Physicians of Edinburgh and a Fellow of the Royal Society of Edinburgh and was a president of the Medical Society of Edinburgh. At the present time he holds the positions of Assistant Physician to the Royal Infirmary, Edinburgh, Lecturer in Clinical Medicine in the University of Edinburgh and Lecturer in Medicine in the School of Medicine of the Royal Colleges at Edinburgh. He served as a member of the Royal Army Medical Corps in the great war from 1915 to 1919 in Mesopotamia, India and France and was awarded the Military Cross. He has had wide experience in medical laboratories both in Europe and on the American continent. Since the war he has been constantly engaged in the prosecution of research with the late Arthur Robertson Cushny and later with Professor Meakins and Professor Murray Lyon. He has published numerous papers between the years 1921 and 1929 and is without doubt an outstanding man in the medical world.

Professor H. R. Dew was born in Melbourne in 1891, graduating Bachelor of Medicine and Bachelor of Surgery in the year 1914. He was admitted as a Fellow of the Royal College of Surgeons in 1920. Dr. Dew was educated at Scotch College, Melbourne, before he entered Ormonde College at the University of Melbourne to commence the medical course. During his undergraduate career he obtained honours in all subjects, including exhibitions in biology, physiology and therapeutics. After graduating in 1914 he served as a resident medical officer of the Melbourne Hospital until April, 1915, when he left on war service. He served with the Royal Army Medical Corps from 1915 to 1919 as a medical officer in France, Palestine and Egypt. In 1916 he was awarded the *Médaille d'Honneur* by the French Government. After his war service in 1919 he returned to London for post-graduate work and attended post-graduate classes at Saint Mary's Hospital and other London hospitals. On his return to Melbourne in 1920 he worked in an honorary capacity at the Walter and Eliza Hall Institute of Research in Pathology and Medicine. In 1924 he was awarded the Jacksonian Prize of the Royal College of Surgeons for an important essay. In 1927 he shared the award of the Syme Research Prize of the University of Melbourne for a published work on hydatid disease. Dr. Dew has contributed many important papers to scientific journals and has published two important works. His record as a surgeon is highly esteemed not only in Australia, but also throughout the whole British Empire. Possibly Professor Dew will leave Australia to visit research institutions overseas before he takes up the duties of his chair in Sydney. He was highly recommended for the position by experts in London and Australia.

Professor H. D. Wright was born in Tasmania in 1891 and entered the University of Tasmania with a classical scholarship, graduating Bachelor of Arts in 1910. In 1916 he obtained the degrees of Bachelor of Medicine and Bachelor of Surgery in the University of Edinburgh with first class honours and medals in anatomy, physiology, pathology and medicine. From March, 1916, to November, 1917, he served with the Royal Army Medical Corps in France as regimental ambulance medical officer. From then until February, 1920, he was stationed in north-western India and east Persia. On returning to Scotland he was appointed Lecturer in the Department of Bacteriology of the University of Edinburgh under the late James Ritchie. He obtained membership of the Royal College of Physicians, Edinburgh, in 1921, and in 1922 was awarded the Lister Research Fellowship of the College. In 1923 he took up an appointment as Assistant Lecturer in Bacteriology in the Medical School of University College Hospital, London. In 1926 he was promoted to the position of Lecturer in the Department and in 1928 he was made Reader in Bacteriology in the University of London, taking charge of the teaching and research work of the Department of Bacteriology. In 1925 he obtained the degree of M.D. of Edinburgh with gold medal and in 1927 that of D.Sc.. For the past three years he has been assistant editor of *The Journal of Pathology and Bacteriology*, a member of the council of the Pathological Society of Great Britain and a vice-president of the pathological section of the British Medical Association. During the past ten years he has published many papers relating to bacteriological science. Professor Wright has been spoken of as a man of wide culture with a keen appreciation of the contribution of laboratory science to medicine. He should prove to be a stimulating director of the new Department of Bacteriology.

Proceedings of the Australian Medical Boards.

TASMANIA.

The undermentioned have been registered under the provisions of *The Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Hay, Arthur, M.B., B.S., 1929 (Univ. Melbourne), New Norfolk.

Story, Bernard Samuel, M.D., C.M. (Manitoba), F.R.C.S. (England), 1897, New Town.

QUEENSLAND.

The undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Cornford, William Henry Hall, M.B., B.S., 1926 (Univ. Melbourne), Normanton.

Fox, Raymond William Sydney, M.B., B.S., 1927 (Univ. Melbourne), Emerald.
Pincus, Fabian Franz, M.B., B.S., 1922 (Univ. Melbourne), Camooweal.
Poole, Gabriel Ruscombe, M.B., B.S., 1924 (Univ. Melbourne), Brisbane.

Honours.

DR. HUGH R. G. POATE has been invested with the insignia of Commander of the Venerable Order of the Hospital of Saint John of Jerusalem and has been awarded the long service medal of the order.

Books Received.

A NEW TREATMENT OF TUBERCULOSIS, by Pierre Hulliger, M.D.; 1930. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 156, with illustrations. Price: 8s. 6d. net.

THE PHYSIOLOGY AND ORAL HYGIENE AND RECENT RESEARCH WITH SPECIAL REFERENCE TO ACCESSORY FOOD FACTORS AND THE INCIDENCE OF DENTAL CARIES, by J. Sim Wallace, D.Sc., M.D., L.D.S.; Second Edition; 1929. London: Baillière, Tindall and Cox. Demy 8vo., pp. 233. Price: 10s. 6d. net.

Diary for the Month.

FEB. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 25.—Queensland Branch, B.M.A.: Obstetrical Section.
FEB. 26.—Victorian Branch, B.M.A.: Council.
FEB. 27.—South Australian Branch, B.M.A.: Branch.
FEB. 28.—Queensland Branch, B.M.A.: Council.
MAR. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 4.—New South Wales Branch, B.M.A.: Ethics Committee.
MAR. 4.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
MAR. 5.—Victorian Branch, B.M.A.: Branch.
MAR. 6.—South Australian Branch, B.M.A.: Council.

Medical Appointments.

Dr. Wilfred Arthur Joseph Brady (B.M.A.) has been appointed Acting Medical Superintendent for the Hospital for the Insane, Ararat, Victoria.

* * *

Dr. Marshall Andrew (B.M.A.) has been appointed Government Medical Officer at Picton, New South Wales.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx, xxi.

ADELAIDE HOSPITAL, SOUTH AUSTRALIA: Honorary Surgeon.
ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Medical Vacancies.
CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Honorary Medical Vacancies.
LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Two Resident Medical Officers (male).
MARYBOROUGH HOSPITALS BOARD, QUEENSLAND: Junior Resident Medical Officer.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Anæsthetist.
TRESILLIAN MOTHERCRAFT TRAINING HOME, PETERSHAM, NEW SOUTH WALES: Junior Honorary Medical Officer (relieving).
WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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